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CLINICAL STUDIES IN VESTIBULAR AND AUDITORY TESTS IN INTRACRANIAL SURGERY* (Second Paper).

The Relationship Between the Vestibular Apparatus and Lesions of the Cerebellum from the Evolutionary and Developmental Standpoint. (An Explanation of the Irregularities of the Vestibular Symptom in Cerebral Lesions.)

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Introductory Note: In this paper, by placing emphasis on the evolutionary growth of the vestibular apparatus and of the nervous system, I have attempted to review the vestibular functional development, and to show the influence of surgical lesions on primitive nervous tissue; to which latter the vestibular apparatus primarily belongs.

Rudimentary attributes underlie all of life's mechanisms as they slowly evolved from the early tropisms, reflexes and automatisms of invertebrate life into the higher faculties[1] which man alone possesses, largely because of his acquirement of speech.

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If we consider the vestibular manifestations from an evolutionary standpoint, we may come to a more rational understanding of the irregularities of vestibular manifestations; for in studying lesions involving the vestibular apparatus we should think, not alone in terms of hyperthetical anatomical tracts (many of which may not be present), but should take the larger viewpoint of evolutionary physiology.

Biological Data of Clinical Significance Pertaining to the Vestibular Apparatus: During the past few years, as the wonders of the vestibular apparatus have dazzled our imaginations, we have too frequently regarded it as highly specialized—on the same plane with speech, which latter possesses very definite "tracts," injury to which produce definite and in some cases irreparable manifestations. "Because the vestibular apparatus is *so new to us* we have regarded it as *new to life;* while in reality it is one of life's oldest attributes."[2]

Plants are stationary; they belong to the soil, but they have a rhythmic ebb and flow, analogous to but different from the rhythmic movement observed in the ovum of a salamander (Coghill).

Plants have no nervous system, but the Indian school in its study of sensitive plants, has demonstrated that plants possess areas of especial sensibility; that they are influenced by injury and by heat; and that they apparently have periods of lessened sensibility (periods of sleep); and also that their reaction to an injury is lessened by anesthetics, by drugs[3].

All plants have static orientation. Although their movements are dictated by the winds, they automatically turn their leaves toward the sun; trees grow upward, not downward; the mechanism of which and the reason for which no one has the slightest idea.

But animal life, in the invertebrate, has movement; it is free[4]. The degree of freedom depending upon its species. And in all species it has orientation. When it is attached to one spot, as in the sponges, it has an otolith in the centre of a vesicle made up of neuro-epithelial cells.

The medusa have many otoliths[5] (auditory vesicles) surrounded by ciliary pavement cells; and connected by nerves to a ganglion; which in turn is connected to muscles.

Thus is formed the first "vestibular (sensorial) arc"[6] (Beaunis); to inform the medusa as to the position of its tentacles and to tell it when to open and close its food catcher.

Primordeal otolithic apparatus have divers forms; the vesicle may be open or closed, with single or multiple otoliths.

The nervous mechanism of the invertebrate thus consists of an end organ, connected by nerve fibres with isolated ganglionic cells.

And here in the domain of orientation, the principle of pressure (pressure by the weight of a heavy substance on ciliary epithelial nerve cells), is established; and this pressure is transformed directly to muscles. And thus the principle of posture in low forms of invertebrates is nothing but a co-ordination of the organism to variations in weight on its end organ.

And later a remarkable thing appears, posture—a function that we are in the habit of attributing to the nervous system—has in the lower vertebrates become a part of the tissues themselves.

For in low forms of invertebrates, there is no central nervous system; each end organ is a king unto himself and thus many parts and many tentacles are constantly being removed; but the animal, or creature, thing, whatever you may call it, goes on living, growing, reproducing and dying.

During this living, the sense of touch is the important sense; the commanding principle. The animal lives by touch, it only turns its tentacles toward food when it is touched by the passing morsel. It is oriented by its sense of touch; by pressure.

If an oyster is detached from its bed; its otolithic apparatus tells it that it is wrong side up and it will successively open its shell and with the help of the waves it will ultimately be turned over; thus using a "lock muscle" voluntarily to correct its position.

Thus in the invertebrate, the principle of touch sensation is applied to orientation and it in turn to posture.

But even in invertebrate life refinements, "specialization," "individuality," "intelligence," "choice by experience," "hereditary," call it what you will, is evident; for shrimps a few hours after moulting "pick up grains of sand and introduce them into their ears to serve as otoliths."[7] (Kreidl.)

And when we examine the invertebrate kingdom, we find the different end organs with their sensory nervous ganglia connected with each other; and thus a primitive central nervous mechanism is established; the involuntary (Langdon Brown), autonomic (Langley)[8], sympathetic (Gaskell) vegetative nervous mechanism for visceral control. It has the anatomic characteristics that the nervous ganglia are situated not far from the receptor; and the different ganglia are connected together on the ventral (under) side of the animal.

And later in life's evolution when other end organs are added, such as the fasceted simple upright retinal eyes of the crabs, they also are attached by ganglionic masses to its autonomic nervous system; and each end organ is adapted to its environment or developed by its environment. For in deep sea crabs, those species which even during their swimming laval stage, have the possibility of expo-

sure to the light, have pigment in their iris and retina, while the eyes of those which are always in darkness, lack pigment. "Light evidently exerts a very important influence on the developmental stage of the eyes of deep sea crabs."[9]

And in all the primitive animals, although their eyes now allow them to see in a very imperfect way, it is their otolith pressure systems that tell their position in space.

If a crab is placed on its back, it will with great difficulty turn over, because its otolith pressure system informs it of this fact, even when its eyes are removed. In other words, it is its pressure apparatus that is the important element in its adaptation to its environment.

And again in the vertebrates in the sea; the fishes. They live and move freely in a media but little different than the weight of their bodies.

But in fishes, life, in its creative evolution[10], has evolved a lateral line system to which the otolith system is now attached.

Bony fish have but one large otolith, and it continues to grow yearly in concentric layers like the "rings" of a tree, so that in certain fishes the age of the fish can be told by its otolith lamina[11]. Frost[12] has collected several thousand varieties and sizes of otoliths.

These organically produced inorganic stones contain the key of many of the anthropological problems; for otoliths are the only existing remains of myriads of fish forms that have been and passed[13].

However, in the vertebrate, in addition to the autonomic nervous centres, there is a central nervous system, now placed on the dorsal (posterior) side of the animal.

And Gaskell[14] thinks that the central nervous system of the vertebrate attained its dorsal position by a "neutralization" of the alimentary system of the invertebrate.

The chief part of the vertebrate's central nervous system—the brain—is situated in the head of the animal; with long commissural nervous fibres extending downward to its tail.

And along with the increase of ganglionic tissue in the cephalic end, the specialized end organs assume a position either intimately attached to the brain, as the vestibular apparatus; or actually an outgrowth of it, as the retina. Each end organ is controlled by the central nervous ganglia of the brain but through the brain at the proper moment, the end organ dominates the whole organism.

In insects the primitive auditory organs, consisting of feather-shaped hairs attached to a thin receptive membrane[15]—for the appreciation of sound and the maintenance of equilibrium—are scattered in different parts of their integument, but in the vertebrate fishes all

acoustic elements become localized in two bilaterally placed areas—the acustiofacial areas of the lateral line—attached to the brain's most rudimentary part, the medulla (myaloencephalon[16]).

For now to enable the fish to perform movements in all planes; to be upside down and to know it, and still move in this position, there is developed a semicircular canal system, which has been evolved out of the more primitive pressure otolithic system.

In the combined semicircular canal and otolith systems, the principle of pressure on nerve endings still dominates; but to impulses originating from the pressure of the weight of otolithic calcium stones, there is now added the refinements of pressure impulses from the weight and movements of the fluid in the semicircular canal and of the displacement of the membranous canals themselves[17].

A new peripheral end organ connected with and a part of the otolith system is thus created for the purpose of allowing the fish to be in any position at will, and to quickly assume this position. This is made possible by the semicircular canal system which is intimately allied with the muscular mechanism, especially of its eyes, but also with all other muscles of its body; and an otolothic system which now is chiefly connected with the mechanism of static position.

It is from experiments on dog fish that it is believed that the otolith system regulates the fishes' static equilibrium and the semicircular canal system in kinetic equilibrium[18].

The equilibrium apparatus in the lateral line organs "respond to slow vibrations" and are sensitive to the pressure of the column of water above it, for slight differences in the weight of the column of water above the fish apparently dictate its position in relation to the vertical. Thus when a jar containing small fish is slowly centrifuged in the horizontal and vertical plane, "their dorsal fins constantly point towards the surface. The fish swim around the periphery against the rotary current induced by the centrifugation."[19]

The fish thus has a primitive semicircular canal system, which has been added to a fully developed otolith system both operating by pressure; the one from the pressure of stones on nerve ends; the other by the action of currents of fluid on nerve ends and the displacement of the nervous tissue of the membranous labyrinth itself. Both systems, otolith and semicircular canal, are attached to a mass of ganglionic cells in the primitive medulla. The semicircular canal system is chiefly connected with the muscles of the eyes and head, so that it may quickly turn in any direction, in any plane, on perceiving or sensing its prey or enemy. For the labyrinth (the combined otolitic and semicircular canal organ) apparently is the most important nervous mechanism in many forms of fish.

In sharks the labyrinth continues to grow during its entire life. It is nearly as large as its brain[20].

And in the lower vertebrate we find that the flexed posture (the basis of orientation) is inherent in the tissues themselves.

Thus the embryo of the amblystoma (laval salamander) assumes a coiled posture before its muscles are attached to the central nervous system at all; the "flexed" and later the "coiled" posture being the primitive muscular tonus positions which uniformly appear prior to the successive S positions necessary for swimming. The amblystoma's "limb is able to respond very precisely to stimuli that arise within the body (proprioceptive) as the result of a particular posture"; and this before the limb responds to stimuli from the outside. "Posture is intrinsically involved from the very beginning with the behavior pattern of the entire animal, for the animal can swim successfully with the nervous system transacted at the level of the auditory vesicle. This means that the mechanism which is essential to the swimming movement is virtually located within the level of the muscle segment."[21]

And while posture is inherent in the muscles themselves, the central portion of this primitive nervous mechanism consists of numerous short neurons. In fact, a direct pathway is not present in primitive mechanisms (such as for orientation). Direct tracts, like the pyramidal, are the latest acquisition to the nervous system. This principle is carried through all the central nervous systems, including man. "The lateral fillet by way of which the vestibular, a low vital (Kappens) or protopathic (Head) mechanism reaches the tectum of the midbrain is such tissue."[22]

Consequently in the nature of the vestibular apparatus, its central mechanism (being of a very primitive nature) cannot consist of centrally located long isolated associative fibres, nor can it clinically be treated as consisting of telephone wires (isolated and definite anatomical tracts), pressure on such or such, causing such and such isolated symptoms. For the body lives, moves, walks, stands as a whole; not until the cortex is developed does real cerebral localization of function begin. And the lower the function the more intimate the association with the central ganglionic masses.

All through vertebrate life in the fishes, sensation is the controlling element. Examination of the retina of fish demonstrates that it does not see in the sense in which we use the term. The visual area of its brain is very small. It perceives an object, especially by its movement. That is why fish are such cannibals; they eat their own.

For it is chiefly by the sense of touch that aquatic animals differentiate stationary things. If you put a piece of cotton in a tank, if the fish does not see it strike the water, it will pass it by even when the fish's body touches the cotton. But if you soak the cotton in oil, as soon as the fish touches the cotton it will snap at it and then, perceiving by taste that it is not food, it will emit it again.

Beebe[23] states that so-called "man-eating sharks" pass unnoticed the diver at the bottom of the sea; that all authentic cases, about nine, of sharks having attacked humans occurred either when the human was swimming or actively dangling his feet in the water. It is apparently the motion of the moving flesh that the shark sees. Also he states that sharks normally do not attack each other, but if a shark is caught on a hook, during the commotion that it makes in its struggle to escape, plus probably the scent of the organic tissue that is liberated, other sharks will attack it and devour it.

The shark's semicircular canal system is open at one end and in the lower fishes the septum consists of vertical canals only: the horizontal canals are a later addition, probably when life evolved an up and down movement at right angles between the head and body by the formation of a neck. For in mammals the plane of the horizontal canal is parallel with the plane in which the animal normally carries its head. Thus the plane of the horizontal canals in sheep and buffalo, which carry their heads low, are at an entirely different angle from their torsos with those of the horse, which carries its head high[24]. It is possible that it is the later addition of the horizontal canals in man that makes him less susceptible to the influence of the cold caloric in the upright position when the intracranial pressure is increased[25].

During the creative evolution of life at the time that the semicircular canal system appears, there also develops in that portion of the primitive medulla which contains the vestibular nuclei, an outgrowth of nervous tissue, the primitive cerebellum. Its function is the co-ordination of all the end organ impulses with the proper muscular movement of the organism.

Thus in the higher fishes we have a combined apparatus (otolith and semicircular canal systems), for the orientation and equilibrium of the body, and especially of the head and the co-ordination of the eyes. The end organ is a specialization of the sense of touch through the application of pressure. This end organ apparatus is connected with the primitive bulbar area of the brain and especially with the co-ordinating organ of the central nervous systems. And now from the lateral line system in the inferior part of the vestibular end organ there develops the primitive legena for the perception of

tones; like the two former also connected with the facioacoustic area in the brain stem, and with a neural extension into the ganglionic cells of the lateral fillet of the brain which are there before there is any cochlear. Fish do not hear in the sense that man does, but they perceive sound—noises—much as we appreciate the roar of a train as it goes through a tunnel.

Thus in the evolution of the nervous system in the lower vertebrates, orientation, equilibrium, posture, muscular tone, hearing, are all one continuous process; so intertwined that they cannot be separated one from the other[26]. Equilibrium, posture, muscular tone because of their low genetic origin are largely taken over by the muscles themselves; head orientation has specialized short neural cerebral ganglionic mass connections in the midbrain, but hearing, because of its later development and greater specialization, has long associative fibres throughout the brain.

And life crawled out upon the land—in the primitive amphibia, of which unfortunately there are no members extant. And in the frogs we find less evidence of the otolith system; already its importance has largely diminished this because the media in which the amphibian moves is different, and it has adjusted itself to its environment, or its environment has altered its structure[27]; perhaps both.

In the frog the well developed semicircular canals have direct connections with the muscles of the legs; its semicircular canal systems are directly in control of its muscular reflexes during active movement. And thus McNally[28], by destroying the proper canal, can influence the posture, tone and the extensor protective movements of the side toward which the frog is tilted as well as the flexion of the opposite side (reciprocal innervation; Sherrington). And these compensatory (protective) movements of the extremities persist up to man[29]; for sudden alterations in the position of the membranous canals cause the throwing forward of the arm and leg of the homolateral side and the extension of the opposite.

And now in the amphibian, we see the development of a reflex to hold the animal right side up and "to quickly right" itself when it is upside down so that it may move with the greatest facility on the land.

A crab crawls on the bottom of the sea, and if the tide suddenly recedes and leaves it on the sand, turned on its back, its locomotion is suspended until it can with great difficulty right itself. It has no righting reflex. But a fish during active motion swims in all planes although in static equilibrium it assumes the ventral position. If you throw a fish into the water and it strikes on its back, it does not

immediately right itself; its abdomen is seen glistening as it darts away to safety. And many fish (the rays and sharks) assume the dorsal position at critical periods; when they "strike" for their food near the surface of the water, but they do not right themselves immediately. It is this ability to move in all planes with equal facility that explains the functional importance and the anatomical large size of the labyrinth in the fish. They perceive in what plane they are, probably from the otoliths, and they move normally and with equal facility in that plane no matter what it may be. This they do by a co-operation of all the muscles, as well as of the fins and the motor oculai. This co-operation is largely through the semicircular canal part of the fishes' vestibular system.

Fish have no neck and in them a righting reflex is unnecessary. But a frog either dives into the water or hops on land, and it has a neck. With difficulty you can throw a frog so that it will land on its back. It has righting reflexes but they are slower than in the terrestrial animals that are liable to fall in the air (cats, rabbits and squirrels), in whom the quick righting reflexes (correction of posture by way of muscular tonus of the neck and limbs under the control of the otoliths and the semicircular canals) is the important thing.

"In rabbits the utricular otoliths are without relation to induced nystagmus either from caloric or rotation, but the utricle and saccule participate in the labyrinthine cervical reflexes; while the semicircular canals are of no importance for these reflexes."[30]

In the amphibia, taste and touch are still the commanding instincts. But then life became terrestrial in the reptiles and all running things, and in them the sense of smell is predominant; and now in the enlarged brain, the rhinoencephalon, the olfactory area is the main portion.

And then the birds, evolving from the reptiles, took to the air and in them is the most completely developed semicircular canal system.

And then terrestrial life took to the trees and immediately the sense of sight assumes the important rôle. In the tree shrew, a tiny lemur which looks straight forward and has stereoscopic vision, the visual area of the brain's new mantle attains an enormous size proportionately; and the proportional size of its rhinoencephalon is diminished[31].

And now creative life modifies and amplifies the orientation and equilibrium apparatus to adapt it to the new media.

And in those quadrupeds that are half aboreal, as in the cat, we have the quick "righting reflexes" of Magnus well developed; a combination of labyrinthine orientation with muscular neck and body

reflex, independent of vision. In the righting reflex the labyrinth plays a commanding part, for in cats and rabbits the only permanent effect of the removal of both labyrinths is seen during falling. Thus normally a cat when blinded falls "free" and lands on its feet. Its otoliths tell it when its body is upside down and its labyrinthine neck reflexes acting on all its extremities—on every muscle in its body—quickly corrects its position. But a monkey does not fall free; in falling it corrects its position by the use of its upper extremities or its tail.

And now the primates in the semiupright position, and at last, man.

Man with frontal lobe developed; temporal axis expanded, and neural associations augmented. Man in the upright position, with upper extremities liberated, with binocular vision, association with fixation, and the presence of a fovia. Man with speech and thus the capacity to transmit his experience. Experience dominates his inhibitions and controls all his acts and conditions all his reflexes[32]. And now in man the control of posture and muscular tone and orientation by the central vestibular apparatus, has become largely lost and its reflexes vestigal. For vestibular destruction in man while it causes nystagmus, vertiginous sensation and deviation of the extremities, all these are very temporary.

In man all the mechanism to preserve equilibrium and posture for life in the heavy media of water are lost.

a. The air bladder, probably to tell the depth of the fish away from the surface, is gone. *b*. The lateral line clefts have become vestigial; and *c*. the otoliths microscopic; *d*. the membranous labyrinth fails to reach the possibilities of its embryorial growth (Werner), as the membranous utricle is comparatively simple.

And with all these anatomical deteriorations comes loss of function; for some of our reflexes, like some of our structures, are vestigial[33].

Thus, if the microscopic otoliths of a fully grown man have any function, it is in static equilibrium. There is no clinical or pathological evidence of disturbances of otoliths except it be in the peculiar sensation and loss of muscular tone that the long osculatory movements of a ship or the long vertical drop of an elevator produce in seasickness[34].

The righting reflexes of Magnus in grown man are absent; but there is reason to believe that in the human fetus the central vestibular mechanism assumes muscular control and "causes the normal head presentation."[35] For the vestibular nerve to the canals is myelonized by the end of the fifth or the beginning of the sixth month

as well as that of the maculae-saccule and utricle. This is about the time that the child assumes the head position in the pregnant woman. Also in infancy, the muscular control of the vestibular apparatus is clinically demonstrable during the first few days after birth and continues during the first year. If an infant be lifted by a hand under its abdomen, it flexes its head and extends its legs so that the back becomes concave. Righting reflexes have been elected in hydrocephalic monsters (Magnus).

The dependence on properly functionating labyrinths with their associated areas in the brain, for the normal acquisition by an infant of crawling, sitting, rising and walking, has been demonstrated by the microscope[36].

After the first year all these reflexes are too vestigial or too obscured to be clinically demonstrable.

The control of the muscles of the extremities by the labyrinth, so pronounced in the frog, is possibly still present in sudden movements, such as in stumbling, but it is now minimal, as a deaf mute with loss of bilateral labyrinth reactions also exhibits a proper extremity correction, undoubtedly from proprioceptic impulses from the muscles, skin, bones and articulations. In fact, the chief efferent impulses in the control of postural tone in man are proprioceptive from the muscles themselves; and so dependent have the different nervous mechanisms become on each other that every part of the nervous system has a part in the nice control of postural reflex[37].

With these anatomical changes and loss of domination by the end organ has occurred profound alterations in the central nervous system. For there is anatomical evidence that in man the reflex tracts of the nervous octavus has not the importance that they have in lower types[38].

Among the anatomical changes the red nucleus, which plays a necessary part in the locomotion in cats[39], in man has lost its commanding size and character[40].

Neurobiotaxis[41], whereby with change of function there is a gradual shifting of the nuclear masses of the central nervous system according to the impulses that pass through them, affects the central vestibular apparatus.

The vestibular nuclei in lower vertebrates lie on the dorsal edges of the medulla; derived from them and connected with them are the nuclei which later become the cochlear nuclei as in reptiles they gradually shift into the cerebellum and in the higher animals lie completely in the cerebellar substance.

All through life's evolution, new "auditory" cerebral tracts develop. In all these there is a vestibular element. Thus there are many auditory association fibres in man which are not present in any animal. But the peripheral nervous fibres still betray the great difference in their phylogenetic development. Thus compression of the vestibular nerve, of very primitive nature, while it causes degeneration of the nerve itself, does not affect the end organ (maculae) or Scarpa's ganglion. On the other hand, compression of the acoustic nerve results in degeneration of both Corti's organ in the labyrinth and of Corti's ganglion on the nerve (Crowe)[42]. Thus the auditory nerve does not follow the Wallerian law; it is more highly specialized than a spinal nerve, to which the vestibular has been likened.

Further, there is anatomcal evidence suggesting that the peripheral ganglionic mass of the vestibular mechanism (Scarpa's ganglia) is even more primitive than the segmental spinal cord ganglia; for the cells of spinal cord ganglia in the embryo are bipolar but later develop into tripolar cells; while the cells of Scarpa's ganglia retain a primitive bipolar character throughout life (Spiegel).

Experimental work on animals must have a limited application, as experimentally used animals, frogs, rabbits, pigeons, are the result of life's creative offshoots that had long left the stem that led up to man; and as all the primitive amphibia and the primitive reptiles are extinct, if we must use analogies from experiments on animals for the problems of orientation, we should use the carnivorous primates, or better still, the man apes—for they are the only species now existing to which man is even distantly related.[43]

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37. Langworthiy, O. R.: The Control of Posture by the Central Nervous System. Physiological Reviews, Vol. 8, No. 2, April, 1928. The author thinks efferent impulses are conducted by (a) the dorsal, (Flechsig) direct cerebellar, or dorsospinal cerebellar tract and the (b) ventro, or lateroventrosuperficial tract (Gowers) and (c) by fibres from the posterior columns (gracilis and cuneatus). All these (a, b, c) terminate in the cerebellum and carry impulses to it from the muscles; which suggests the importance of the cerebellum in the control of posture. The remaining fibres carrying proprioceptive sensitivity, travel in the median lemniscus to the thalmus and to the cortex. The vernis sends excitatory or inhibitory impulses to the final common path, by three efferent systems: (a) reticulospinal, (b) vestibulospinal, and (c) rubro-spinal. All go to make the common final path.
38. Winkler states that on account of the development in man of the corticopontocerebellar system and of the influence of this system on movements and of the development of the corticospinal tract, the reflex system of the nervous octavus has not the importance in man that it has in the lower types. Deiter's (1) spinal tract; (2) the spinal portion of the two longitudinal fascicili; (3) the rubrospinal tract; and (4) all the paths designated as accessory pyramidal tracts, have a relatively feeble development in man.
39. Windle, W. F.: The Relation of the Level of Transaction of the Brain Stem to the Occurrence of Decerebrate Rigidity in Young Animals. *Jour. Comparative Neurology.*, Vol. 48, No. 2, Aug., 1929.
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CORRECTION.

Attention is called to the fact that in the April, 1931, issue of The Laryngoscope the legends for illustrations for Dr. L. Guggenheim's article on "Centers of Ossification in the Head," page 235, were transposed. The legend for the lateral view appeared under the verticomental view, and vice versa.

COMPARATIVE SURGICAL ANATOMY OF INFANT, CHILD AND YOUNG ADULT TEMPORAL BONES AND MASTOID PROCESSES (LANTERN SLIDES AND SPECIMENS).*

Dr. Philip S. Stout, Philadelphia.

The temporal bone has always been something of an enigma. Its development, position in the skull, relationship and contents are extremely interesting but we can only touch on a few highlights, tracing from the embryo to the adult bone, showing particularly some of the variations of the air cell development in the mastoid process.

The embryonic head skeleton, according to Piersol, Aray, Miller *et. al.*, includes: *1.* Brain case. *2.* Capsular investment of sense organ. *3.* Branchial arches. These arches are the counterparts of the arches supporting the mouth and pharynx of fishes and tailed amphibia. The first and second arches are concerned in the development of the temporal bone and its appendages.

From the first arch develops malleus and incus.

From the second arch develops stapes, also tympanohyal and stylohyal processes, which fuse to form the styloid process.

Petrous portion and mastoid portion are intracartilaginous development.

Squamous portion and tympanic portion are intramembranous development.

Ossification begins as early as the eighth week of embryonic growth. Three or more centers of ossification in the periotitic capsule unite into a single center from which the whole cartilage is transformed into the petrous and mastoid portions.

The mastoid process development begins after birth by an outward bulging of a part of the petrous bone. The mastoid cells result from evagination in the surrounding bone of the epithelial lining of the middle ear and antrum, mostly, of course, from the antrum, which is quite large at birth, at times even larger than in the adult bone. Incidentally, in infants the drumhead, ossicles, oval and round windows, cochlea, semicircular canals, aquaductii cochlea and vestibuli, fossae endolymphaticus and hiatus fallopii are nearly as large as in adults.

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*Read before the Philadelphia Laryngological Society, March 3, 1931.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication April 2, 1931.

Fig 1. Six months fetus showing labyrinth.

Fig. 2. Newborn, showing antrum and air cells.

Fig. 3. Newborn; opaque substance in antrum.

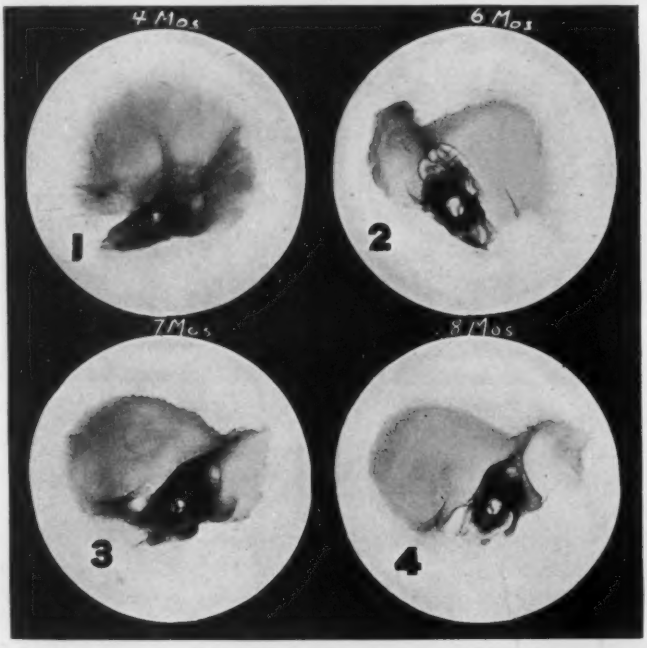

Fig. 4. 4-6-7-8 months infant, showing development of air cells and antrum.

In infancy the temporal bone is easily separated into its component parts; the petrous, tympanic and squamous. As is well known, the infant's temporal bone is surgically quite different from that of the adult (see pictures). The tympanic antrum, usually spoken of as the mastoid antrum, is higher in the infant than in the adult. It may

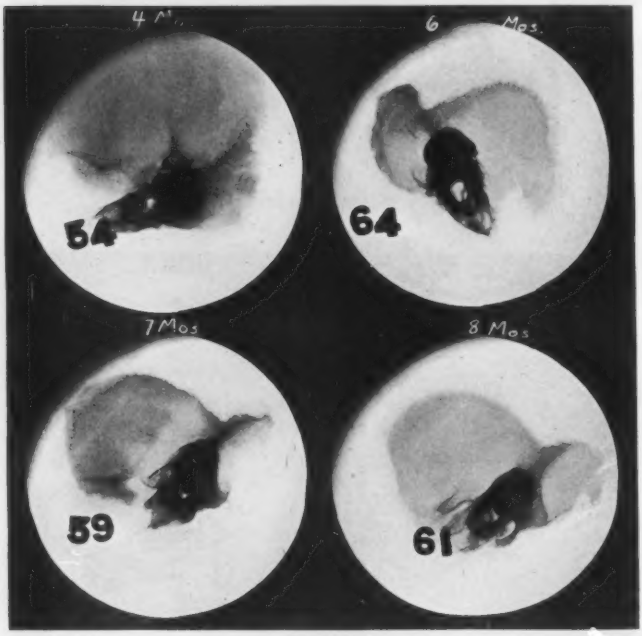

Fig. 5. 4-6-7-8 months infant; opaque substance in antrum and air cells.

be above the linea temporalis, if that structure can be made out. In fact, the antrum is above and posterior rather than directly posterior to the external canal. Failure to recognize this fact has led to much confusion in finding the antrum in a nonsuppurative case. The linea temporalis, as stated above, is not always easy to make out in the

infant and young child. The spine of Henle may not be present or may be difficult to make out, although you can nearly always make out the posterior bony prominence of the end of the annulus tympanicus. This annulus is flat in infancy but increases in outward growth, trumpet-like to one-half-inch or more. This lengthens the external

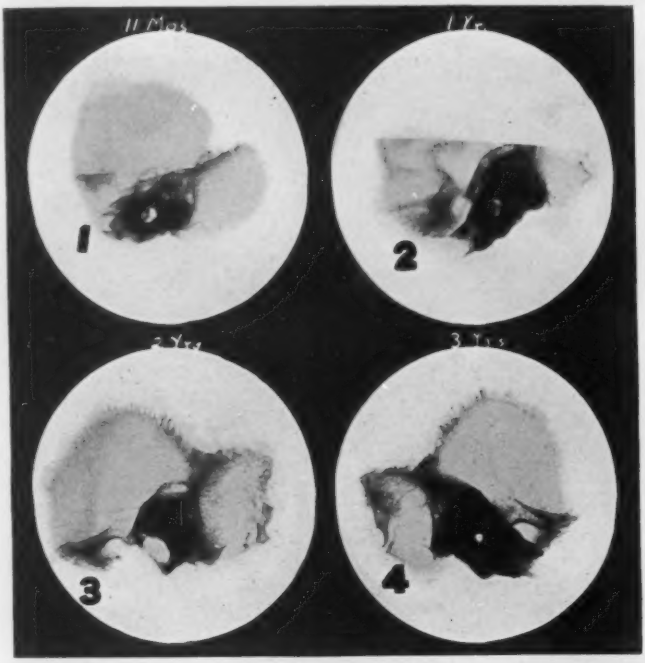

Fig. 6. 11 months, 1-2-3 years child, showing development of antrum and air cells.

auditory canal, changes the whole contour of the external appearance of the temporal bone, changes the relative position of the exit of the facial nerve, which in infancy is on the surface of the bone just posterior to the external canal, while in adults it is one-half of an inch or more from the surface of the bone in the stylomastoid foramen.

The infant and young children mastoids show an antrum rather large (see pictures), with sometimes a few cells surrounding it and some diploe, seldom sclerosis; while in the older, sclerosis is not infrequent, diploe rare, 2 per cent, and, of course, a large increase in areation as the age increases.

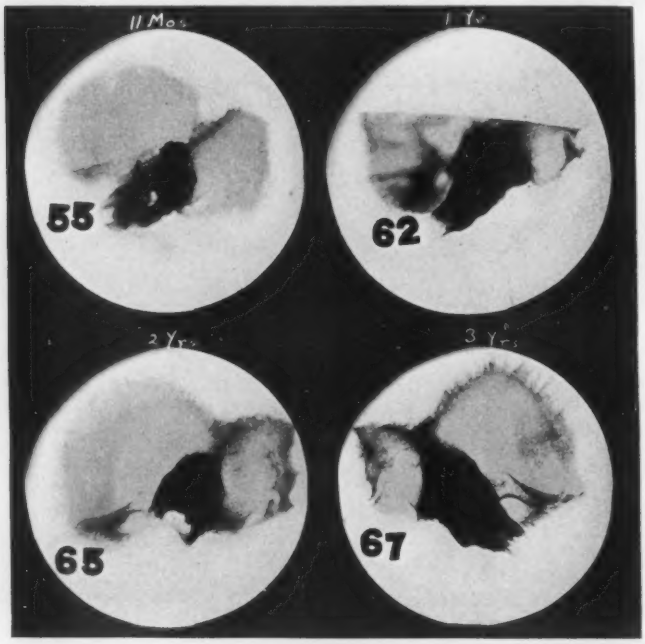

Fig. 7. 11 months, 1-2-3 years child, showing opaque substance in antrum and air cells.

In the older child the pneumatic cells extend toward the tip of the mastoid. So-called tip cells are nearly always present; however, there is not the marked extension of cells posteriorly as in the adult.

The sigmoid sinus in infants is seldom the seat of inflammation because of the few cells near the sigmoid. Squamous covering of

the infant bone over the antrum is not quite fused to the mastoid and therefore pus finds an easy exit; also the bony covering is very thin.

The development of the mastoid process is gradual, but with many exceptions and variations caused by the arrested development, shape

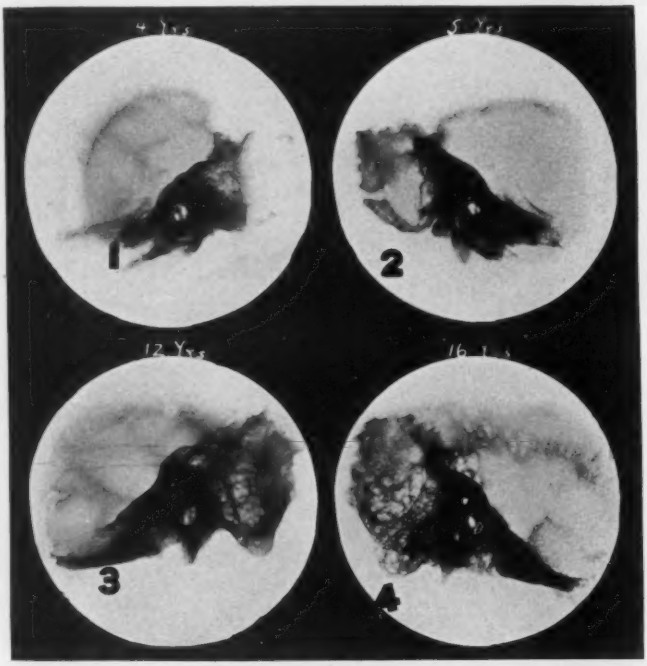

Fig. 8. 4-5-12-16 years, showing development of antrum and air cells.

of head, character of air cell development, and disease, such as chronic otitis media causing sclerosis.

A word in regard to the collecting of specimens of infant and children's temporal bones. They are difficult to obtain. The reason for this is obvious. For a number of years now I have collected here

and there young temporal bones; one a year of a different age would be a good average. My interest was first directed to the collecting of temporal bones by my good friend, Dr. E. B. Gleason, who showed me his collection of temporal bones back in 1911. Incidentally, he and Dr. Alexander Randall have done as much on the

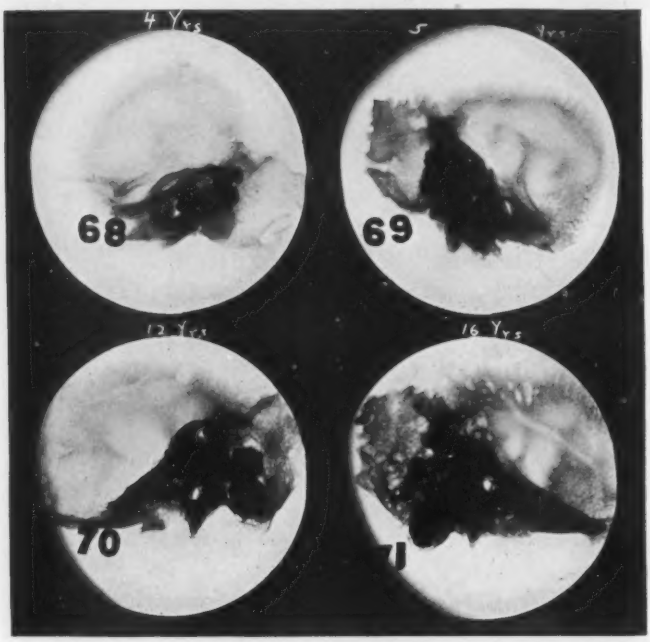

Fig. 9. 4-5-12-16 years, showing opaque substance in antrum and air cells.

study of the temporal bone as anyone in America. In 1922 I got a few specimens in Europe. A few years ago Dr. O. V. Batson, of the University of Pennsylvania Graduate School, who was making a trip through Europe, was good enough to gather, at my request, some 16 specimens of infant and young children temporal bones.

These made a very valuable addition to the collection. These bones are among those shown in the pictures.

For the newborn specimen I am indebted to Dr. Parson Shaeffer, of the Baugh Institute of Anatomy, of Jefferson.

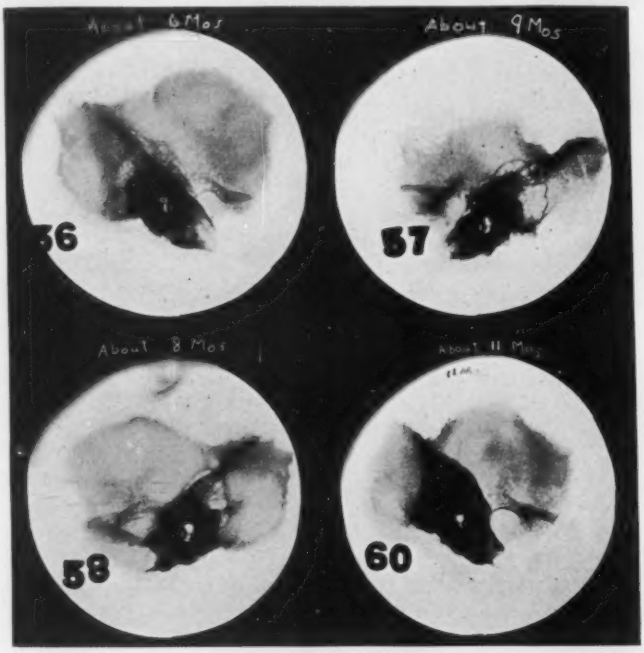

Fig. 10. 6-8-9-11 months infant, showing unusual development.

I am greatly indebted to Dr. R. Manges Smith, of Jefferson, for his hearty co-operation in making the X-rays of these temporal bones, and also to Dr. William Manges for his co-operation and permission for the use of his department.

269 S. 19 Street.

SECONDARY SIMPLE MASTOIDECTOMY WITH CHOKED DISCS AND SEVERE CEREBRAL SYMPTOMS: CASE REPORT.*

DR. H. M. SCHEER, New York.

April 11, 1928: N. L., Manhattan Eye, Ear and Throat Hospital.

Family History: Irrelevant.

Past History: Tonsils and adenoids removed four years ago. Two and one-half months ago, following acute purulent middle ear infections, subsequent to measles, double simple mastoidectomy was performed. Uneventful recovery. Patient discharged three weeks ago. Both middle ears dry after one week, and mastoids healed in seven weeks.

Present History: Purulent discharge from left ear for one week, with earache.

Physical Examination: White male child, age 7 years, well nourished and developed. Heart and lungs negative. Throat clear. Right canal, drum and mastoid negative. Old scar over right mastoid process. Left canal shows profuse mucopurulent discharge. Pinna stands away from head. Edema and redness behind left ear, in region of old scar, severely tender to touch and fluctuating.

Diagnosis: Acute otitis media purulenta; left secondary mastoiditis. Admitted to Manhattan Eye, Ear and Throat Hospital on April 13, 1928. Temperature, 102°. Secondary simple mastoidectomy performed. Granulations and pus present in the mastoid cavity were removed. Dural plate over the middle fossa was soft necessitating small exposure of dura at tegmen antri. Temperature following operation rose to 103.2°. Patient apparently comfortable for 48 hours after operation. While walking through the ward, on the second day after operation, I noticed that the patient appeared to be stuporous and did not respond properly. Complete dressing done at this time showed profuse purulent yellow discharge in the mastoid cavity and profusely soaking up the entire dressing. The wound was immediately laid widely open. Within 30 minutes from the onset of the stupor the child began to have localized twitchings, first on the right side of the face, then the right arm and leg, then the left arm and leg, and before another half-hour had elapsed he was

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in generalized convulsions, which came with such rapidity that by the time he was removed to the operating room he was practically in clonic contracture, which was so severe that respiration was impaired and death was impending. The operating room was prepared in greatest speed. The patient was held under anesthesia by chloroform to control his convulsions and in this way allowing him to breathe. After rapid cleansing of the wound and thorough antisepsis with 2 per cent mercurochrome, a posterior incision was made to enlarge the wound. An area of dura was exposed, about the size of one's palm, in the region of the middle fossa, also uncovering the lateral sinus through its entire extent. The posterior angle between sinus and dura was entirely removed and the cerebellar dura exposed posterior to the sinus and internal to it. The dura in the region where exposure had been made at the time of secondary mastoidectomy appeared covered with dirty gray granulations. The entire wound was thoroughly cleaned and after proper antisepsis of same, at the suggestion of Dr. John R. Page, upon whose service this case was admitted at the hospital and who was present at the operation, ten punctures of the brain were made with the brain forceps; each from 2 to 4 c.m. in depth, without finding pus. Dura was not under severe tension. A small incision was made in the dura each time puncture was made. Similarly, puncture was made into the cerebellum without finding pus.

Wound was cleansed with alcohol, followed by 2 per cent mercurochrome, and kept widely opened. Upon reaction from the anesthetic following the operation, the patient was rational and no evidence of any convulsive twitch recurred. Temperature following operation had reached normal by fifth day, with the exception of several rises between the sixth and seventh days postoperative. Temperature then stayed normal until the thirty-sixth day postoperative, when it gradually rose to 104°, during which time the patient developed a rash, which was diagnosed as measles, for which he was quarantined. After his temperature was normal for ten days following this condition, he was discharged from the hospital.

The interesting features during the postoperative period were those referable to the eye ground examinations, which Dr. Farley, of Manhattan Eye, Ear and Throat Hospital, was good enough to keep following very regularly from the day of operation, April 13. Some of his reports are as follows:

April 16: Slight optic neuritis, right eye.

May 11: Slight papilloedema O. U. No hemorrhage seen, vessels dilated, veins in O. D. particularly.

May 12: Optic neuritis more marked, about 3 diopeters of swelling. No paralysis of eye muscles, although there was distinct strabismus of the right eye.

May 15: Papilloedema a trifle more marked.

May 21: Papilloedema much more marked, each eye about 5 diopters of swelling. Marked venous engorgement O. U., with one small hemorrhage, temporal side of the disc O. S.

May 23: Papilloedema possibly more marked than last examination. Very small hemorrhage in right eye. Several in the left eye.

June 1: Papilloedema greatly reduced O. U., about 3 diopters of swelling, venous engorgement much less, and no hemorrhages seen in either fundus. Very marked improvement.

June 4: Two diopters of swelling of each disc. From this time on the eye grounds assumed normal appearance.

X-ray examination of the nasal sinuses on May 25, by Dr. F. M. Law, reports: The left ethmoid has moderate involvement, due to granulations or exudate; right shows mild involvement from thickened membrane. The frontals are absent, and the antra and sphenoids are clear.

Report of X-ray of the skull on May 7 by Dr. Law is as follows: The left mastoid area gives the appearance of either bone destruction in the temporal bone above the mastoid, or surgical removal of bone. There is no evidence of brain abscess observed at this time. This removal is very extensive.

Repeated neurological examinations between the dates of April 19 and May 23, by Dr. E. S. Zabriskie, revealed nothing of any pathological significance except what was already mentioned regarding the eye examinations. Lumbar puncture on April 16, just before the cerebral exploration, yielded 27 c.c. of clear fluid under fair amount of pressure; examination of fluid showed moderate trace of globulin, moderate trace of sugar, trace of albumin, moderate trace of lactic acid, 11 small lypmhocytes per c.m.m., no bacteria found on direct spreads. Culture shows no growth.

Blood findings were as follows: April 17, blood count: White blood cells, 20,000; polymorphonuclears, 80 per cent; small lymphocytes, 20 per cent. April 26: Hemoglobin, 68 per cent; red blood cells, 3,495,000; white blood cells, 10,800; polymorphonuclears, 60 per cent; small lymphocytes, 30 per cent; large lymphocytes, 10 per cent. May 23: Red blood cells, 3,560,000; white blood cells, 13,000; polymorphonuclears, 72 per cent; small lymphocytes, 20 per cent; large lymphocytes, 8 per cent.

May 23, lumbar puncture yielded 12 c.c. of clear fluid under normal pressure, very faint trace of albumin, no globulin, heavy trace of sugar, very faint trace of lactic acid. Seven small lymphocytes per c.m.m. Culture shows no growth. May 26, lumbar puncture yielded 8 c.c. of clear fluid, eight lymphocytes per c.m.m.; otherwise examination was negative.

Repeated urine examinations were negative.

From the facts as stated in the case there was unquestionable cerebral irritation, followed by increased intracranial pressure; although no pus was found at the time of the cerebral exploration.

1. The possibility of brain abscess still occurred to us. Some with whom the case was discussed, following its cure, felt that such brain abscess might have been absorbed.

2. Coma, complicating a toxic nephritis, was to be considered, but was eliminated because of the negative urinary findings.

3. Diffuse cerebral meningitis was eliminated because of presence of negative cerebrospinal fluid findings. There was undoubtedly localized meningitis present, but this in itself would not cause the train of symptoms and physical signs as occurred.

4. Cerebral edema, with its resultant increased intracranial pressure following severe toxemia or following the trauma due to the punctures into the brain and such incessant convulsions as occurred in this case was the diagnosis ultimately arrived at. The cause of the very severe convulsive seizure was in all probability the marked toxemia and absorption from a very badly infected operative wound.

The case is presented with the hope of bringing about some discussion from the floor. In this manner the correct diagnosis may be placed onto the case.

Dr. F. M. Law demonstrated lantern slides of this case.

On date of reading this proof, May 5, 1931, the patient is perfectly well.

522 West End Avenue.

AN ANALYSIS OF ONE HUNDRED CASES OF DEAFNESS.

Dr. E. Martin Freund, Albany, N. Y.; and
Dr. Harold Hays, New York.

In making an analytical survey of this nonselected group of hard-of-hearing cases, one finds many interesting situations. The object of this study was not to describe any particular form of treatment but to marshal a group of facts from which an otologist might deduce a logical approach to the proper diagnosis of his deafened patients. It is our belief that similar studies will increase the general knowledge concerning deafness and thus prove ultimately of benefit to those so handicapped.

The cases in this group were studied under the following headings: Sex, age, duration, of condition, past history, heredity, occupation, chief complaints, previous treatments, contributory factors, progress of the disease, coincidental affections, and physical findings in the ear, nose and throat.

Sex does not seem to be an important factor. In this series of one hundred cases, indiscriminately selected from the private practice of one of us (H. H.), the proportion was: males, 42; females, 59. We do not consider this of any special significance, except that pregnancy may be a minor factor in predisposing the mother to deafness if there are any latent otosclerotic cell rests present. But this is by no means an original claim on our part. It is only a conclusion arrived at after digesting the general opinion of leading otologists.

As we approach the fifteenth year of life we begin to find the incidence of deafness more evident, and from then on till the thirty-fifth year we find the "high incidence age" of deafness. That is to say that from 60 to 70 per cent of the cases of deafness under treatment are between these ages. This does not exclude younger children. It merely means that a young child may go along for several years with progressively defective hearing before he or his parents become aware of the affliction. Then it may be several years before any attention is paid to it or an attempt is made towards its correction. More than 50 per cent of people who were deafened gave a history of a "running ear" dating back to an early age. Age was

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also an important factor between the twentieth and thirty-fifth years. It is in that period that the respiratory and nasal mucous membrane diseases often involve the Eustachian tubes, eardrums, inner ears and mastoid cells. This period shows the greatest incidence of impaired hearing, but this is also the period in which the treatment of cases produces the most encouraging results. From the fortieth year upward, vascular changes in the inner ear as well as ossicular changes and thickening of the membrana tympani should be considered.

Heredity seems to play a very minor role in deafness, except in luetic cases and in otosclerosis. Incidentally, recent observations have shown the tendency to consider otosclerosis as of obscure syphilitic origin even if the diagnosis cannot be definitely established.

Occupation was not of great significance except in distinct cases calling for constant and increased atmospheric air bombardment of the eardrums, such as occurs in boiler-making, aviation, riveting, artillery firing and underground or subsea work. Spontaneous rupture of the eardrum, hemorrhage into the cochlea and even sudden complete destruction have been reported as a result of the above occupations.

It was a significant fact that a large number of the cases here investigated came not so much because of their deafness but rather to seek relief from their tinnitus. This latter complaint ranged from a mild to a severe degree. Also the disturbing sense of fullness in the ears and head were most depressing to many of these patients. A number of them were quite content to go along through life with defective hearing so long as the tinnitus and fullness in the head could be relieved or controlled. Tinnitus was noted to follow in the wake of diminution of hearing in from one to five years. It occasionally preceded deafness itself. Often the sudden discovery of deafness in an adult resulted in a tendency for drastic measures and treatment. These patients would in alarm and desperation shop for an otologist who would give them the best prognosis. Here it may be wise to caution the specialist not to be too enthusiastic in his promises. After all, when deafness is well established, with a distinct chronic pathologic process, the best one can hope for is to arrest the progress of the disease. The patient must be made to understand this. He must be distinctly informed that the function of hearing cannot be fully restored and that an attempt will be made to preserve the residual percentage of hearing, and if possible to improve the existing condition after a reasonable and judicious observation and treatment. Excessive Politzerization or undue stimulation of

the drum may also temporarily exaggerate the deafness present or in some cases even do some harm; and in that manner drive the patient on to the next otologist. One of our cases had consulted ten ear specialists previous to her coming to our office, claiming that she was rendered worse by the various treatments that may have been too energetic.

In glancing through the previous histories of these cases, we found some very interesting data. Aside from the various and irrelevant diseases, there were certain elements that were constantly encountered as associated or predisposing factors in producing or resulting in deafness. At least 60 per cent of this series of cases gave the history of repeated "head colds" of various years' standing. Over 70 per cent of these cases presented a distinct history of one or more attacks of tonsil and adenoid infections. More than 75 per cent of the latter occurred in the first and second decade. In the instances where no tonsillectomy had been performed, the diseased glands were easily demonstrable.

A history of deafness in the family was not an outstanding fact. Less than 40 per cent of the patients had a distinct or a vague recollection of some member or members of the family so afflicted. In most of these cases it was usually a sister or grandmother that was mentioned. We question whether this can be of any special significance. However, the female members of a family were in predominance in the ratio of two to one. Deaf mutism was demonstrable in the histories of two cases.

Postnasal drip was another complaint often stated by the patient. This occurred in about 35 per cent of the cases and resulted no doubt from faulty sinus drainage and chronic nasopharyngeal infections.

A few (8 per cent) of the cases traced their deafness as following febrile and other constitutional diseases such as rheumatism, typhoid and scarlet fever, diphtheria, pneumonia and influenza during and after the adolescent period. A fairly large number of the patients gave a positive history of "running ears" when young. Unfortunately this latter condition occurred during infancy or early childhood and no accurate data could be ascertained. It would have been interesting to note whether these acute otitis media cases had had myringotomies performed or whether the drum was permitted to rupture spontaneously.

Some cases showed a range of deafness of from three weeks to 30 years before consulting us. But inasmuch as these long standing cases did not come under our observation primarily, this discrepancy

in time cannot be interpreted with any degree of certainty or as leading to a distinct conclusion.

Another fairly constant complaint (about 50 per cent) was that some intercurrent diseases tended to emphasize or aggravate the deafness present. Among the previous operations indicated, the following had been performed: Tonsillectomy and adenoidectomy, 30 per cent; nasal septum resections, 10 per cent; myringotomy, 10 per cent; mastoidectomy, 5 per cent; turbinectomy, 5 per cent, and maxillary antrotomy, 2 per cent. A significant relationship between progressive deafness and faulty nose-blowing was very apparent. Nearly every case that complained of "ears puffing out when the nose was blown" showed progressive diminution of hearing. Where the nose was blown gently and the nostrils not completely blocked during the act the patients almost invariably grew no worse in their loss of hearing. This brings out the fact so forcefully emphasized by one of us (H. H.) in his textbook, that forceful or faulty nose-blowing is a factor in producing or aggravating deafness.

Associated also with the deafness were some of the following diseases, in their respective frequency: Ocular disturbances, infected teeth, stomach and intestinal diseases, hypertension, rheumatism, lues, general debility and hay fever.

This brings us to the physical findings gleaned on examination of these cases. Here we find a wide range of conditions, though a few are outstanding and fairly constant. For the purpose of convenience these pathologic findings may be classified according to the location, with the emphasis stressed in the numerical occurrence. More than 65 per cent of the cases showed retracted drums, varying from a slight to a marked degree. The lustre of these membrana was usually pale or colorless, the landmarks often being completely obliterated. On the other hand, more than half the number of these retracted drums were fairly movable (relaxed); less than 10 per cent being excessively rigid, the remainder being moderately fixed.

The next most frequent pathology was found to be present in the tonsils. Over 50 per cent of the cases showed evidence of tonsillar disease ranging from chronic hypertrophy to the small fibrous submerged type. Frequently these tonsils gave no evidence of active symptoms, while in many instances it was possible to express a purulent or cheesy content without the presence of constitutional signs of absorption. A moment's digression may be permitted to point out the necessity of removing even a slightly diseased tonsil in all cases of deafness. For there is no doubt of the liberation of toxins that are continuously manufactured within the gland sub-

stance. The proximity of these glands to the opening of the Eustachian tube and thus into the middle ear presents a constant source of infection and irritation to the auditory tract.

The next pathologic location was evidenced in the nose and its accessory sinuses. The inferior and middle turbinates were found diseased in about 20 per cent of the cases, the posterior tips especially of the middle turbinates often showed hypertrophy and polyp formation, thus causing interference with drainage from the ethmoid and sphenoid sinuses. Septal deviations to one or both sides and septal spurs were frequently encountered, but in only 5 per cent of the cases was there evidence of real nasal block.

In several cases we found a marked increase in lymphatic tissue in the nasopharynx and it was not uncommon to find a generalized edema of the nasal mucosa. As to the role of the septal deviations one cannot interpret them as actual causative factors. Certainly though, they play a contributory part in the continuation or aggravation of the condition by interfering with proper nasal ventilation. Transillumination in this series disclosed no striking findings. Actually only two cases of frontal sinusitis and three of antrum opacity were found.

The fossa of Rosenmüller was often seen to be the seat of adhesions. As this fossa is situated directly behind the Eustachian promontory, the patency of the tube was greatly interfered with, limiting the proper air communication to the inner ear from a mild to a marked degree. The breaking up of these adhesions frequently produced marked relief and continued to show considerable improvement in the patient's hearing so long as the adhesions were kept from reforming.

Diminished tubal patency was next considered. Undoubtedly the patency of this channel plays an important rôle in the deafness of extra-aural origin. Quite a number of the cases showed interference with this opening, due to the previously mentioned conditions as well as polypoid growths at the mouth of the tube. These polypi were readily discerned by means of the electric rhinopharyngoscope. There were 12 cases of Eustachian tube stenosis, 8 of polypi at the opening of the tube, and 12 of general edema of the tube along its tract.

Among the remaining physical defects not so frequently noted were thickened eardrums, remnants of tonsillar and adenoid tissue, extensive perforations of the drum, atrophy of the drum, atrophic rhinitis, eczema of the auditory canal, calcareous deposits on the drum and cholesteatoma.

These latter physical findings were usually of several years duration and can be considered as chronic factors in producing the existing deafness. Some cases showed a multiplicity of conditions, though not a constant relationship. One condition, however, proved rather striking, and that was the usual coexistence of tinnitus with retracted and partly fixed eardrums. In this connection a study of the vestibular reactions might have brought out additional worth while facts. The mechanism of tinnitus is still theoretical, though we have no doubt that within the near future that problem will be at least partly solved.

In our office we used the 2A audiometer exclusively as it gives uniform and more accurate results. The type of deafness as affecting bone conduction and air conduction was fairly equally distributed, except that in the younger patients it was nearly entirely confined to interference with the bone-conducting apparatus. This latter statement, of course, does not include several of the cases where there was definite history of toxic neuritis of the auditory nerve or complete destruction thereof following diphtheria or scarlet fever.

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SINUSITIS FROM THE VIEWPOINT OF AN OSTEOMYELITIS.*

Dr. G. B. Trible, Washington, D. C.

An increasing amount of study has been paid to the bone involvements of the sinuses, and is now being extended to the temporal bone as well, and with this better knowledge of the processes involved, the relationship of the various types of infections, the modifications occurring due to the differing development and anatomical types, a more rational and comprehensive plan of treatment may be the result[1].

In 1927 a presentation of ideas along this line, with this title, was made before the Detroit meeting of the American Academy of Ophthalmology and Oto-Laryngology, and further experience has tended to confirm these beliefs and further pathological work has demonstrated that in many cases the lack of healing must be due to invasions of the osseous structures.

The early work of Hajek, followed by Sluder and Skillern in this country, and later by McMahon, is constantly being added to, and a recent article by Hayman[2] presents the continental view and shows a tendency to include what were considered minor pathological findings under the term of osteitis. He feels that there is really no difference between osteitis and osteomyelitis, that the terms denote merely a difference in the localizations and that osteomyelitis should not be too much restricted, but should be applied, not only to processes that are complicated with septic manifestations, but also in cases in which sepsis is absent, but in which a process has a progressive tendency and extends over a long period of time.

When we consider the anatomy of the cell-bearing bones of the head, it can be seen that they are fundamentally flat bones and that the inflammatory changes that take place must be somewhat analogous to such changes in other parts of the body.

There is one difference which will be considered later, and that is the allergistic sinusitis, which apparently rarely in the early stages does invade the osseous structure, though invasion of the deeper layers of the mucosa characterized by an excess of eosinophiles is pathognomonic. Whether or not the theories of calcium deficiency in allergistic cases are correct and tend to make more rapidly possible

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the later inroads on the osseous structures is a point that remains to be worked out, and any way so far, it is merely our province to record the clinical findings. There is, of course, the great triad, sinusitis, allergistic phenomena, such as hyperesthetic rhinitis, or hay fever, and the more pronounced bronchial asthma, all presenting phenomena somewhat similar in blood pictures and not constant in relation to the subjective symptoms.

Reviewing the embryology and histology, we know that in the flat bones of the skull, the osseous tissue is laid down in the mesenchyme, an embryonic connective tissue, lying between the brain and the skin. This mesenchyme becomes more richly vascularized and active proliferation of the cells takes place, the characteristic osteoblasts appear, and grouped off, become the first bone cells; later there follows a deposit of bone salts. The first bone is spongy, is resorbed and deposited to permit growth of the brain. This destruction and resorption is carried out by the osteoclasts and is a perfectly natural process, which goes on until death. Some of the air cells, as the antrum, for instance, continue the resorption until the tooth roots may project into the antra in later life, when they do not so project previously.

The mechanism of infection of the nasal sinuses, so far as it relates to those of nasal origin is briefly, an invasion of micro-organisms from an extension of an existing rhinitis or nasopharyngitis. The mucosa swells, becomes edematous, the ostia are obstructed, secretions stagnate, cilia are lost, and the natural current from the periphery to the ostium is interrupted, and destruction of the superficial epithelial layers may occur. The defenses are the phagocytic action of the epithelium, the hyperemia that occurs then, follows, later the accumulation and the extrusion of the leukocytes, with their destructive action on the bacteria, and then the escape of the accumulated secretion through the reopened ostia, or by bursting through at some other point. In the chronic or recurrent condition, there are found both areas of scar tissue formation and atrophy associated with the other extreme, the marked hyperplasia of the mucosa, with later polypoid degeneration and in many instances a combination of these forms of pathologic changes of the mucosa. In fact, it is rare that a single type of pathology occurs throughout. With deeper invasions, the weak mucoperiosteum is involved by extension, and in the chronic cases there is a persistent infection of the subepithelial layers and the presence of bacteria can be demonstrated in sections, while in still deeper invasions, there is found involvement of the bone, with proliferation of bacteria in the blood spaces, which may

produce changes that will lead to superficial necrosis and death of the part.

There is still a tendency to consider as osteomyelitis only the massive changes, such as occasionally follow surgery, and it is probable that this view persists because of our early medical education, and that our early ideas of osteomyelitis are those of the long bones, such as the tibia and femur. In the long bones, however, there is an actual channel filled with marrow, which only blends into the cancellous structure at the joints, and suppurations involving, as they often do, the bone marrow, are massive and frequently characterized by the speedy loss of the entire shaft of the bone.

Pathological changes in the flat bones cannot be viewed in the same light as those in the long bones, and bone pathology here must be considered somewhat as that presented in the ilium or scapula, which do resemble it very much in the gross infections, but even here there is some difference, for the bony elements are circumscribed and protected by muscles and superficial tissues, while in the sinuses we have simply long, thin rows of cancellous tissue between compact layers of cortex protected only by mucous membrane and a very superficially situated and weak mucoperiosteum, that is, in the parts exposed to the air. It readily can be seen that such a condition exists nowhere else, with the exception of the Eustachian tube and middle ear, and strangely enough in most infections of these structures, the tendency is for nature to replace destroyed bone by the addition of bone salts and the eburnation of the remaining structures, while in the flat bones bearing the nasal accessory air cells, the tendency is rather more often to the rarefaction of bone and resorption of the calcium salts.

We must further divide the osteomyelitis of the sinuses into the localized or circumscribed varieties with very little tendency to spread, and which will heal, if the focus is removed, free drainage established and the reparative processes favored, and into the diffuse or spreading osteomyelitis, in which spontaneous healing does not take place. From a review of the recent literature, from our cultures and from hearing several papers along this line in the last year or so, some of which have not appeared in publication, the impression has been gained that the bacterial agent responsible for these cases is usually the staphylococcus in contradistinction to the streptococcic infections. Whether the streptococci do not find such a favorable field for growth in the bone, or whether those organisms cause such overwhelming infections that they escape the bony boundaries and invade the cranial contents before local lesions have been developed is a question yet to be answered.

The mechanism of invasion in either long or flat bones is about the same, though infections of the blood or lymph channel directly through to the medullary structure are more frequent in long bones. Second, a local infection in the mucosa which may spread through the periosteum and then to the bone is found, or a direct inoculation by trauma and in this connection would probably appear the cases following operative intervention. It is likely that in these postoperative cases a too liberal exposure of the bone by extensive elevation of the periosteum may contribute to susceptibility, by lessening or interfering with the blood supply.

Infection in bone extends along the Haversian canals, the calcium salts are slowly absorbed, the infection spreads, leaving islands of bone between areas of infection. If a piece is surrounded and cut off, it sequestrates, giving a different picture in the flat bone than in the long one.

In cartilage the infections start from the periphery and not from the center, as in bone, and in either bone or cartilage, if invasion and destruction are not much more rapid than regeneration, very little disturbance in the bony economy results. As mentioned in the other paper, these slow bone changes are made use of by the orthodontists, who move teeth in the young, merely depending upon pressure to rarefy the compressed bone which is absorbed by the osteoclasts and the carbonic acid content of the blood and new deposits fill in the spaces back of the teeth.

The classic picture of an osteomyelitis is that of a disease, inflammatory in origin, involving the bone, arising in the case of the long bones most frequently in the medullary tissues and in the flat bones from extensions through the periosteum. This is particularly the case in the air spaces, for there we find no true periosteum, merely a mucoperiosteum. The infection, under these circumstances, is that arising from a superficial necrosis, directly under an eroded portion of the muco-periosteum, or by an island of infection around a nutrient or perforating vessel or by extension of a thrombophlebitis from an infection arising superficially.

All the sinuses have in common this loosely attached mucoperiosteum, but the power of resistance to an osteomyelitis varies. In the frontals the sinus margins are extremely susceptible, as are the walls of the nasofrontal ducts. In the latter case this area is most dependent, so the tendency would be for secretions to accumulate and the mucosa become edematous. The frontals seem to be the most susceptible to surgically caused cases of extensive osteomyelitis, especially the frontal plates, and this probably comes from the disturb-

ance of nutrition caused by the elevation and stripping away of the periosteum.

In the sphenoid, the mucoperiosteum is more firmly attached and less often exhibits osteomyelitic changes. In the ethmoid, there is apparently less evident tendency to direct necrosis, but more frequently there is produced a low grade inflammation, which has been studied for several years in great detail by McMahon, who came to these conclusions: That there were five marked changes in the bone, rarefactions, necrosis, osteoporosis, fibrosis and hyperostosis, and he felt that the inflammatory changes in the walls of the blood vessels diminish the supply of blood to the part, causing the production of connective tissue.

The antrum shows in practically all reports the greatest number of conditions involved requiring surgery, but relatively few reports of massive necrosis. It would seem that there must be a selective resistance, for with the great complication of extension of disease from the teeth, granulomas, softening and rarefying the bone or pushing up the periosteum, abscesses of the tooth apices, which may burst into the antrum, or which may be spread by simple extension, or through a thrombophlebitis, there is still a paucity of bone necroses of the massive type. This is all the more to be wondered at when we consider the trauma in skillful, let alone unskillful extractions, with the elevations of periosteum and denuding of bone, under conditions far from surgically perfect.

The widespread incidence of allergistic conditions give them an importance, even without their continued presence leading to the development of a secondary sinusitis of a purulent type. Among these allergistic phenomena there are the various titles of vasomotor hyperesthetic rhinitis, or hay fever, bronchial asthma, catarrhal rhinitis and sinusitis, with production of nasal polypi preceded by edema of the mucosa lining the sinuses. Later the characteristic rhinitis with thickening over the ethmoid and sphenoid, as has been brought out by Hansel[3], who believes that the vasomotor, or hyperesthetic rhinitis is the early stage, while the hyperplastic rhinitis or sinusitis, or rhinosinusitis is the advanced stage in a current infection, and the establishment of a chronic infection, with the added unfavorable condition relative to drainage and aeration, ends by erosion of the mucosa, invasions of the periosteum, and infections of the bone with the probability of lack of cure, particularly from operative measures alone. These cases are all the more confusing in that they give positive evidence of a surgical sinus infection with our present methods of diagnosis, such as transillumination, the X-ray and the use of differentiating Roentgen-opaque solutions, which still show the pres-

sure of vastly thickened mucosa, filling defects of all types, projecting polypi, or cyst formations, the usual criteria relied upon to establish a diagnosis.

These allergistic phenomena must modify our conception of sinus surgery to a great extent. The reported papers in the recent symposium on allergy at the Fifty-second Annual Congress of the American Laryngological Association as well as the work of Sewall and his followers in San Franciso, has brought this to the fore in sinus therapy. The finding of an absolutely blocked nose with sinuses filled up and then in a few days finding nothing, with again a recurrence of the same phenomena presenting all the diagnostic points of sinus occlusion and infection, dark on transillumination, giving an X-ray finding of increased density, pus on aspiration and after filling the cavity with Roentgen-opaque solution, the discovery of a thickly proliferated wall and then in a few days no symptoms, and but little on operation, if performed, tends to give pause to any operator.

Massive osteomyelitis, a most distressing, as well as serious complication of an otherwise good operation, is most serious in all cases, fatal in many, limited to the frontals for the most part; occurring in this region as an inflammation over a wide area, producing abscess of the bone and destroying tissue[4], but less often found in the other sinuses, rarely in the other air cell-bearing bone—the temporal. In the frontal, the osteomyelitic area may involve either the anterior perpendicular plate or the posterior plate. The ones of the posterior plate are naturally less often recognized, for there is frequently a meningitis and the condition is considered as a meningitis, or brain abscess, with the origin not traced.

As illustrative of the different types of osteomyelitis, the following cases are reported:

Mr. G. H., age about 50 years, the first case reported below, is that of an acute gangrenous periosteitis developing in the left antrum and frontal, probably on a basis of an old infection of the antrum, and it presented a most interesting clinical picture at operation. The mucosa was absolutely black in both the antrum and the frontal, and foul smelling; grossly, appeared similar in texture and color to the stewed fat of terrapin. The acute symptoms appeared very suddenly, intense headache, edema and swelling around the left eye, elevation of temperature, intense pain, foul smelling nasal discharge. The pathological report follows:

"Sections show Haversian canals near the periosteum containing numerous pus cells and exudate of serum and fibrin. The periosteum itself shows advanced necrotic changes and considerable fibrin throughout. The free surface of the periosteum is covered over by

a fibrinopurulent exudate in which we find very large numbers of pus cells, and a moderate number of red cells.

"The microscopic picture is that of a very active acute inflammatory process.

"Diagnosis: Acute osteitis with gangrenous periostitis."

The patient made an uninterrupted recovery, the frontal wound was left wide open. It was considered probable that a secondary operation would be necessary, but it healed very quickly, and so far no secondary surgery has been required.

The second type illustrates the chronic type involving the frontal on a basis of an acute infection which produced at the same time an orbital abscess.

M. E., child age 10 years when first seen. On examination it was doubtful if any operative procedure would be worth while. She was unconscious, temperature ranging between 105° and 106°. Face, neck and body covered with a scarlatinal form eruption. Left side of face swollen, eye closed, nose showed purulent secretion. An immediate X-ray showed cloudy left antrum, ethmoid and frontal. Under light, general anesthesia, a rapid exposure and drainage of the left antrum, and removal of the anterior end of the left middle turbinate was performed, and a few days later an incision and drainage was made, using the orbital portion of the Killian to drain the orbital abscess. An erosion was found in the frontal through the orbital frontal plate. After the acute condition had subsided, the frontal was opened and drained, and about a year later there was a recurrence of swelling around the left eye, of the boggy edematous type, and the frontal was opened June 15, 1929, at Georgetown Hospital, and a large sequestrum removed from the floor of the frontal. The pathological report was as follows:

"Secretions from the decalcified bone show evidence of a chronic osteitis, apparently of some standing. Many of the Haversian systems are somewhat atrophied and in a few instances some of the Haversian canals appear to be completely occluded from secondary laminations; many of the secondary laminations have calcareous deposits. Many of the lacunae are quite large and dilated, contain dead bone cells or are completely vacuolated. Examination from a section of the periphy seems to indicate that a periostitis is also present, although no periosteum is attached. No evidence of any malignant change is present."

This cleared up very rapidly and there were no symptoms for about a year, when there was a recurrence of swelling, accompanied by a superficial abscess along the line of incision, which was opened by Dr. Gatwood, of Richmond, as an emergency measure, and she

was referred back to this office, where after a week's treatment, with no result, the incision was reopened with general anesthesia and another sequestrum found, and at the same operation, a more extensive removal was made of the anterior frontal palate. Pathological report follows:

"Chronic osteitis. Chronic osteomyelitis. Periostitis. The piece of tissue labeled No. 2 is a thin, flat piece of bone, rather compact in character, which on microscopic examination exhibits more or less diffuse necrotic changes. Sections show a well defined compact bony structure, the outlines of the lamellae and canaliculi being ill defined,

with numerous passages representing lacunae which are vacuolated; these contain an occasional dead bone cell. Toward the periphery the bone seems to be somewhat more compact than it is in the central part; it is stained slightly deeper and does not exhibit the same type of degenerative change seen in the central portion. The picture presented is that of a chronic osteitis possibly associated with a chronic periostitis.

"Sections from piece of tissue labeled No. 1 show a mass of cancellous bone, together with small portion of the periosteum. The trabeculae are somewhat irregular in contour and shape, showing considerable atypical formation of the lamellae with peculiar serpentine lines of calcification containing lacunae of varying sizes in which bone cells can be demonstrated, but many are vacuolated in a great many instances, the bone cells are apparently dead. The inter-

trabeculated spaces show low grade proliferation of the delicate stroma, together with some patchy cellular infiltration; well defined endothelial proliferation of many of the smaller vessels is noted. The microscopic picture presented is that of a low grade chronic osteomyelitis of long standing."

These reports are more or less similar to other reports in the series and are the only ones given for that reason.

The photograph showing the antrum with the fistula leading to the bone was one of the few cases of extensive necrosis of the antrum which we have seen in this office. There was a bilateral Luc-Caldwell and on operation there were areas of necrosis on the right side. Fistulous formations followed on that side which did not clear up and it was considered unwise to do any further operative pro-

cedure. He gave, in this case, a positive Wassermann, with no history or evidence otherwise of luetic involvement. He has been given extensive therapeutic tests, but apparently has shown no results as far as his bone condition is concerned, though his Wassermann has become negative.

The photograph in the other case is one which evidently had an osteomyelitis of the nasal and frontal bones. He was operated on in numerous hospitals and by some of the most capable operators in the country, and was finally sent to the southwest, for it was considered any further operative procedure would be of no value. At the time seen there was a purulent nasal discharge, as well as several small fistulas in the wound area.

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RETROPHARYNGEAL ABSCESS COMPLICATING TONSILLECTOMY WITH EXTERNAL APPROACH.*

DR. SAMUEL SELTZ, Cincinnati.

Retropharyngeal abscess occurs most commonly in children between the ages of 2 and 5 years and is usually secondary to acute upper respiratory infections, otitis media, acute or chronic sinusitis, tonsillitis and the exanthemata. I have been able to find very few cases reported in the literature following tonsillectomy: Whether this is due to their infrequent occurrence or the failing of the laryngologists to report them, I do not know.

There are two lymph glands on the retropharyngeal space at the level of the atlas which drain the nasal fossae and their accessory sinuses, nasopharynx, Eustachian tube and middle ear. The efferent vessels of the retropharyngeal glands empty into the deep cervical lymph chain. Since retropharyngeal glands do not drain the tonsils these can infect the retropharyngeal glands only indirectly through involvement of neighboring pharynx.

The first stage of the infection is an adenitis or periadenitis of the retropharyngeal glands, followed in 24 to 48 hours by suppuration. Frequently resolution takes place before suppuration occurs.

The abscess may: *1.* Burst spontaneously and drain into the larynx and lungs, occasionally causing asphyxia and death or producing multiple abscesses of lungs. *2.* The pus may dissect its way along the lateral wall of the neck and point in the postcervical triangle behind the sternomastoid muscle. *3.* Burrow downward in the retropharyngeal space and point into the anterior cervical triangle anterior to the sternomastoid muscle or dissect along the prevertebral fascia and extend into the posterior mediastinum. Fortunately the latter is very infrequent.

Incision of the abscess in the pharynx is the commonly accepted method of treating the average case and in the great majority of cases is successful, but where the abscess is very extensive and there is evidence of pointing outward or downward toward the mediastinum, the external drainage should be employed without hesitation. The commonly accepted incision for external drainage is always along the anterior border of the sternomastoid muscle. If one looks at the anatomy of this region, it can be readily seen that incision behind

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the sternomastoid muscle is a much more direct approach without coming in contact with the carotid sheath as one does in the anterior approach.

The etiology of this abscess was probably the carrying of infectious material from the tonsil into the peritonsillar area during the anesthetizing procedure. Therefore one should be very careful when anesthetizing not to push the needle into the tonsil itself. Since this patient had very frequently recurring sore throats he may have had a latent adenitis which was lit up by the tonsillectomy, resulting in abscess formation.

Case Report: D. D., white, male, age 28 years, had a local tonstillectomy done on Dec. 8, 1930. There was considerable bleeding

Fig. 1. Before operation.

from the left lingual tonsillar region, which was controlled by a hemostat. I saw the patient at home on the tenth and he had a little oozing from the left tonsillar bed, which was controlled by local applications of adrenalin. I saw him again on the twelfth and he complained of stiffness of the neck, difficulty in swallowing and general weakness. Temperature, 99.2°; pulse, 96; respiration, 20. The left tonsillar bed was filled with a dirty gray exudate and there was no bleeding. The right fossa was perfectly clean.

I sent him into the Jewish Hospital, where his W. B. C. was 14,200 and R. B. C. 3,500,000, his temperature 99.4°, pulse 96. A culture from left fossa showed short-chained streptococci. He was given hot throat irrigations, local infra-red treatment and the usual sedatives. His neck became stiffer but he seemed to swallow some better. Examination of the pharynx and neck showed nothing remarkable.

On the sixteenth his temperature was up to 103° and his neck was extremely rigid; he was having excruciating pain on the slightest movement. He was also having a great deal of difficulty in swallowing now and examination of the pharynx showed that the entire front wall of the pharynx was pushed forward until it was very close behind the soft palate. Palpation revealed definite uniform fluctuation across the entire width of the pharynx and down as far as one could feel with a finger.

In order to determine the exact extent of the abscess a lateral X-ray of the pharynx was advised and this showed a huge abscess extending well down past the level of the larynx. It was decided to drain it externally due to its tremendous size.

The patient was given 6 gr. of sodium amytal one-half hour prior to operation and under procain injection, incision along the pos-

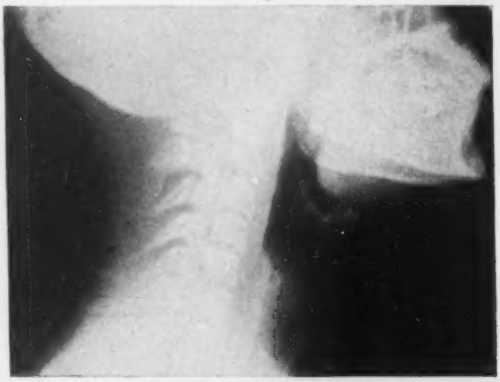

Fig. 2. After operation.

terior border of the sternomastoid muscle was made and with blunt dissection the abscess in the retropharyngeal space was opened at about the level of the cricoid cartilage and a very profuse foul smelling pus exuded from the cavity. After considerable suction a large rubber drain was inserted into the abscess cavity and sutured to the skin.

The wound drained very profusely for about five days, when the tube was removed and a gauze drain inserted. His temperature had now dropped to normal and he was discharged on Dec. 26. The wound subsequently closed to the surface by second intention and the patient is now perfectly well.

The accompanying X-rays show the abscess before and after drainage.

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LARYNGEAL ABSCESS.*

Dr. R. E. Howard, Cincinnati.

In reviewing medical literature one finds that laryngeal abscess is an extremely uncommon condition. There are less than 50 cases reported in the literature, but undoubtedly there are many cases undiagnosed because of the difficulty in examining the larynx when this condition is present.

The first case on record was reported by Roland, of Parma, in the thirteenth century. Other cases were reported by Cayol, Latham and Boyle in the fifteenth and sixteenth centuries, but were indefinite as to the cause of the abscess. Laennec reported two postmortem laryngeal abscess cases, in which the cartilages were not involved. Richards, in recent years, collected a group of 26 cases, in addition to the 13 reported by Mackenzie.

There are several theories as to the etiology of primary laryngeal abscess. According to Ziemssen this process is a phlegmonous inflammation of the submucous connective tissue of the larynx, which may begin in the perichondrium, from trauma, overexertion of laryngeal muscles, or catarrhal inflammation, terminating in localized pus formation.

Laryngeal abscesses have developed from stab wounds, deep lacerations and fractures of the laryngeal cartilages, as well as cauterization with acids or alkalies which have been accidental or taken with suicidal intent. A few laryngeal abscesses have been caused by gases or hot vapors, or as a result of injury to the tissue by a foreign body.

Secondary laryngeal abscess may develop from an extension downward into the laryngeal cavity of an infection of the nasopharynx, tonsillar or peritonsillar region. These conditions also arise in the course of measles, scarlet fever, or develop from a pyemia, erysipelas or general sepsis. One occasionally sees these cases in conjunction with tuberculosis or syphilis of the larynx, as a secondary condition to the specific degenerative changes already present.

The abscess may be intra- or extra-laryngeal, unilateral, bilateral or in the commissures. The intralaryngeal origin is generally in the thyroid cartilage or epiglottis, while the extra laryngeal origin is on the anterior surface of the epiglottis, aryepiglottic folds, or the pyriform fossae.

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Records show that out of the 26 cases collected by Richards, ranging in age from 9 months to 80 years, that the majority of cases are within the 20 to 30-year period.

The symptoms of this condition are fairly uniform and constant. The first symptoms may be a low and hoarse cough with some difficulty in swallowing. The process is gradual, may cause a cervical adenitis, and in the intralaryngeal cases difficulty in respiration. The relation of treatment and mortality is quite striking in reviewing 25 cases. In ten cases where the abscess was not opened, nine died, while in ten cases where it was evacuated, only one died. Out of five cases where spontaneous rupture of abscess occurred, only one died.

With this brief history and description of laryngeal abscess, I would like to report three cases of laryngeal abscess.

Case 1: M. K., age 33 years, a single woman, was admitted to the Contagious Hospital, May 17, 1928, from Longview Hospital, with the diagnosis of scarlet fever. Patient was mentally unstable and a veronal addict, and had been in this mental institution for varying periods in the past three years. Approximately one year before admission to the General Hospital for scarlet fever, patient was examined during an attack of acute laryngitis and no masses were seen, but there was a slight edema of arytenoids and an intense congestion of the mucous membrane of the larynx. This condition cleared, and as the patient was a singer, she continued her voice training without further difficulty, until a week previous to admission at the General Hospital, when she developed a hoarseness. On admission, there was no difficulty in breathing, but on May 19, 1928, patient became cyanotic and in distress, due to respiratory stridor. There was a marked retraction of supraclavicular space and infrasternal notch, but on auscultation there was an equal amount of air entering each side of the chest. Inspection of the chest showed limited expansion, but equal and rapid. Percussion note was normal throughout, while breath sounds were loud and harsh, due to laryngeal obstruction.

Indirect laryngoscopy revealed a mass involving the left laryngeal region above the plica vocalis and practically occluding the larynx, except for a narrow crescent-shaped slit, through which the patient endeavored to carry on respiration. The mass was acutely inflamed and appeared firm, involving the epiglottis, causing it to be three times its normal size, and extending along the left aryepiglottic fold posteriorly to the arytenoid, and inferiorly to the plica ventricularis. The patient's condition grew progressively worse, so that a low

tracheotomy was performed with difficulty, due to the marked infiltration and induration of the prelaryngeal and pretracheal tissues. The patient immediately breathed comfortably following insertion of the tube, and two days later there was a spontaneous rupture and evacuation of the pus. On indirect laryngoscopy the mass seemed to have disappeared and on the seventh day the tube was removed and the patient could breathe through the larynx.

Case 2: B. N., age 19 years, an undergraduate nurse, developed a sore throat and severe headache on May 21, 1928, and was admitted to Contagious Hospital as a scarlet fever suspect. On May 23 the typical scarlet rash appeared, being first noted on arms, but spreading rapidly over chest and abdomen. Patient then complained of marked sore throat and pain in joints and ran a constant temperature of 102°. May 24, there were enlarged and tender cervical glands, especially at the angle of the jaw. The temperature remained approximately 102°. On the seventeenth day of her scarlet fever, a tenderness developed in the right cervical region anterior and posterior to the sternocleidomastoid muscle, accompanied with stiffness of neck and difficulty in deglutition. Two days later, June 10, indirect laryngeal examination revealed marked edema and inflammation of epiglottis and right aryepiglottic fold, with swelling of right arytenoid. The following day, the patient expectorated a large amount of purulent, blood-stained secretion after a severe coughing attack, and then the cervical symptoms subsided. Laryngeal examination showed a point of spontaneous rupture of the abscess in the middle part of the aryepiglottic fold, which promptly healed.

Case 3: J. S., age 32 years, a colored man, was admitted to Cincinnati General Hospital on Dec. 15, 1928, with a history of a cold for two months. On Dec. 14 he developed a sore throat and experienced difficulty in deglutition and respiration. On examination, patient was found to have a purulent sinus infection and edema of the uvula and soft palate.

Digital examination of the hypopharynx revealed a soft, fluctuant mass located at base of tongue, probably attached to epiglottis.

Indirect laryngoscopy then substantiated the digital examination, in that the large mass at the base of the tongue was attached to the epiglottis. The mass appeared inflamed, and was approximately the size of an English walnut.

On *direct laryngoscopy* the aperture of the larnyx and pyriform sinus was seen to be occluded by a large, ball-like mass, which on palpation with a probe was seen to be fluctuant. The mass, under direct vision, was incised with a peritonsillar knife, and a tablespoon-

ful of pure yellow, creamy pus was evacuated. This one incision proved not enough for adequate drainage, so several scarifications were made in the vertical plane and both pus and blood poured into the throat and was promptly aspirated. The organism found was a short-chain streptococcus. A Roentgenogram showed clearly the extent of this abscess in the hypopharynx. On Dec. 31, 1928, the larynx was last inspected and found to be normal except for slight thickening and scarification of epiglottis.

CONCLUSIONS.

1. The first two cases of this series are of interest, in that the laryngeal abscess developed on the seventeenth day of scarlet fever.

2. In both cases spontaneous rupture occurred, although in the first one, tracheotomy was necessary for relief of respiratory distress.

3. In the third case, with principally epiglottic involvement, incision and drainage of abscess was instituted rather than tracheotomy for respiratory distress.

4. In all three cases the offending organism was the short-chain streptococcus.

2600 Union Central Building.

AN INSTRUMENT, FOR TEACHING PURPOSES, TO DEMONSTRATE THE DIRECTION OF ENDOLYMPH FLOW IN THE SEMICIRCULAR CANALS DURING ROTATION TESTS.*

DR. M. VALENTINE MILLER, Philadelphia.

Recognizing the difficulties of students in visualizing the direction of endolymph flow in the semicircular canals during the rotation tests, and very vividly recalling my own bewilderment at the same stage, it occured to me that an instrument could be devised which would show clearly what takes place in the canals during these tests. The instrument which I here present is by no means ideal but will, I feel sure, greatly help in making the reactions more clear.

As we all know, the semicircular canals are not closed tubes, but communicate at each end with a common chamber called the utricle, the vertical canals on each side entering the utricle by a common crus. The ampullae of the horizontal (or external) canals and those of the anterior vertical (or superior) are anterior, and the ampullae of the posterior vertical canals are posterior. For the purpose of this experiment I have deemed it best to fashion the canals in my instrument as closed tubes with the ampulla appearing in one end as a small bulb, the other end being plugged with cotton. The endolymph is represented by metal beads which, upon turning and sudden stopping, will gravitate in the direction of turning, thus showing the movement of endolymph toward or away from the ampullae.

The instrument is so constructed that the models of the canals are at each end of a crossbar in the center of which is a doll's head to show the relative position of the canals as regards the position of the patient's head. This bar is attached to a tube by a universal joint and this tube, in turn, caps and turns upon a rigid metal supporting rod. The canals and the head, therefore, may be placed in any desired position and so stimulate any pair of canals: 30° forward for the horizontal canals, 90° forward for the posterior vertical canals, and 60° backward for the anterior vertical canals. The head may be bent forward 45° and inclined to the right shoulder for the right posterior and left anterior vertical canals, and 45° forward and

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inclined to the left shoulder for the left posterior and the right anterior canals.

With this instrument, and bearing in mind the five following rules, one may, without difficulty, work out the normal responses:

1. When the canals of both sides are stimulated the endolymph current is toward the ampulla on one side and away from it on the other. The canal in which the current is toward the ampulla receives

Fig. 1. Model of semicircular canals.

the greater (two-thirds) stimulation and so it is this canal which determines the reaction.

2. The slow component of the nystagmus is in the direction of endolymph flow. (Nystagmus named from quick component.)

3. Past pointing is in the direction of endolymph flow.

4. Falling is in the direction of endolymph flow.

5. Vertigo is in direction opposite to endolymph flow.

114 W. Phil-Ellena Street.

LOCAL ANESTHESIA TONSILLECTOMY.

Dr. J. B. H. Waring, Cincinnati.

With present day perfection of local anesthesia technique, it would seem there was little excuse for subjection of the average adult tonsillectomy patient to the discomforts and added risks of general anesthesia. Yet it is safe to say that the vast majority of tonsil operators still insist upon general anesthesia for all tonsillectomy patients, adults as well as children. One reason for the present tonsillar electrocoagulation wave over the country is that it is a local anesthesia "office" treatment technique; bloodless; no hospitalization required; patient ambulant throughout course of treatment; etc.

Wonderful technique that it was in The Master's hands, the Sluder technique is difficult of mastery by the average operator; and it is hardly a technique applicable to all types of tonsils. Likewise it is not so satisfactory with local anesthesia operation; at least, a great many Sluder operators rarely attempt the operation under local anesthesia.

The vast majority of local anesthesia tonsillectomies today are by the so-called snare and dissection technique. It is at least a technique applicable to all types of tonsils, but taking a country-wide cross-section of dissection tonsillectomies in hands of average operators it is anything but an entirely satisfactory technique; in fact, it is estimated that three out of every four tonsillectomies today are imperfect in one way or another. The more experienced and skillful the operator the greater his percentage of perfect tonsillectomies, whatever the technique he employs.

Jacobs and Trauner say this: "Theoretically, tonsillectomy by preliminary dissection aims to remove the entire tonsil intact, with the least amount of hemorrhage. This it often fails to do.

"The capsule is often so intimately adherent to the underlying aponeurosis that it is impossible to separate the two by means of a knife. What usually results is that the knife gets into the fascia back of the capsule and opens many bleeders, or else it gets into the tonsil tissue proper, so that the fibrous capsule is left in the fossa."

That is a perfect pen picture of dissection technique; yet how many dissection operators will admit this? We have the school of sharp dissection, and the school of blunt dissection; with adherents perhaps equally divided. Some tonsils are easy with sharp dissection;

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and some are easy with blunt dissection. I have seen apparently perfect fingernail tonsillectomies; a "modification," as it were, of blunt dissection. A year or so ago an operator read a paper on "My Method" of bloodless blunt dissection. Essentially, this consisted in jamming a heavy curved blade forceps down back of the tonsil; clamping; and slicing more or less of the tonsil off front of clamped blades with scissors. It was bloodless and it was blunt; quite so.

A recent very interesting tonsil paper is one by Neivert on "Pitfalls of Local Tonsillectomy." Dr. Neivert is an adherent of the sharp dissection school. I quote:

"When we have finished and are truly critical of our work and review the various steps, certain factors should come to our mind, which, if improved upon or overcome, *should make the operation safer and less tiresome for the patient and easier and more satisfying to the operator.*

"Make the operation safer and less tiresome for the patient and easier and more satisfying to the operator."

That then is a goal for which all of us can earnestly strive. Yet a Chicago dissection operator lamented to me in this wise:

"I have been doing tonsil work (dissection) for 25 years, and I cannot do a decent job yet. One operation is very satisfactory; and maybe the next is a bloody mess that makes me ashamed of myself."

I think that describes dissection in hands of the operator of average ability; some dissections are very easy, rapid, clean, practically bloodless. Others, and many others, are just the opposite. Unsatisfactory to the patient and still more unsatisfactory to the conscientious operator.

I quote again from Dr. Neivert:

"Our greatest difficulty is with tonsils bound down by scars and adhesions. Here a little patience and a sharp knife cutting close to capsule will keep us from buttonholing the pillars and pulling away chunks of muscle which cause excessive bleeding, possible infection and subsequent scarring. * * How to make sure that one is in the proper line of cleavage is at times difficult. * * Small tabs of tonsil or capsule with tonsil-bearing cells must not remain. * * Taking off the pillars or uvula is scarcely excusable. * *

"Perhaps the thing that worries us most is the bleeding."

Another true picture, which I quote but in part. Can we eliminate; get away from these several pitfalls which Dr. Neivert so graphically describes? Can we make the (tonsil) operation *safer* and *less tiresome* for the patient, and *easier* and *more satisfying* to the operator? The answer is affirmative. It can be done; but with the

average patient under local anesthesia and operated by the average tonsil surgeon, I do not believe it can be done with present day dissection technique. I may be accused of being a one-method operator; so overly enthusiastic as to be blind to obvious difficulties and disadvantages perceived instantly by the cold, critical "show me" observation of operators employing a different technique. Not so. No one is more coldly critical of my own tonsil work than I am myself. For years I have studied closely every tonsillectomy I performed; and if possible I would like to study the tonsil work of every skillful tonsil man. To compare; to evaluate; to criticize; to improve in any way possible.

I am not a one-method operator. Far from it. Every adult tonsil patient is given the choice of local anesthesia tonsillectomy by the suction methods, or by local anesthesia electrocoagulation. General anesthesia tonsillectomy is not employed in adults by me unless the patient insists (and there is no contraindication), or unless the patient is so obviously neurotic and uncontrollable as to make local anesthesia operation impracticable. Such cases are few and far between with proper handling. With the aged and infirm, and with those of defective blood-clotting history and conditions, I regard electrocoagulation as the technique of choice; and in general my results, cosmetic and otherwise, are just as perfect with the coagulation technique as with the suction. The advantages and disadvantages of the two techniques are explained to the patient, "one big sore throat" for a few days and a soft diet, or a "number of little sore throats" and your usual diet and avocation over several weeks' treatment. No hospitalization in either case, unless desired by the patient.

When we can remove the average adult's tonsils under local anesthesia cleanly, rapidly and practically without bleeding, in from 10 to 15 seconds' time per tonsil, without pain, without fright, with clean fossae, pillars and uvula intact, and convalescence shortened by half, and with a minimum of postoperative pain and discomfort, it must be admitted the suction method is at least worth a casual observation by every operator who wishes to make the operation *safer* and *less tiresome* for the patient, and *easier* and *more satisfying* to the operator.

Perfect equipment and perfect technique are essential. You cannot look for perfect suction tonsillectomy work without these, any more than you can expect perfection in any other line of endeavor with haphazard methods.

Compared to the average dissection, rapidity of this suction technique is somewhat astonishing. Not that rapidity is our goal, but

when perfection can be attained with an inherent rapidity, this is not to be overlooked. In a charitable tonsil-adenoid clinic held last fall in the south, some 55 tonsil-adenoid cases were operated by the writer in a morning and afternoon session, working in the crude and hastily improvised operating room of a large rural school; and in a morning session some 25 children were operated between 9 a. m. and 11 a. m. Of course, this was all general anesthesia work and patients were anesthetized and practically ready for immediate operation when placed on the operating table by shifts of anesthetists and assistants, but this should give some idea of the rapidity of the local anesthesia operation also.

Local anesthesia tonsillectomy by the suction technique will be briefly described:

Patient is instructed to eat usual breakfast. A half-hour prior to operation 3 gr. of phenobarbital are given by mouth. When seated upright in operating chair with headrest, the operation is quickly outlined to patient so that nothing frightens the patient or takes him or her by surprise. The patient is told there will be no pain, but is not told the operation will be bloodless. On the contrary, he or she is told that when the tonsil is removed there may be a little blood-tinged saliva with each tonsil, or perhaps a little free blood, but not to worry, that there will be no hemorrhage or any free bleeding to bother about. Fear of hemorrhage seems to disturb patients chiefly, and next fear of being hurt. The patient is assured there will be no pain; that when the tonsil is removed not to cough or clear the throat but to simply lean forward in chair with mouth open slightly and to breathe quietly, so that any saliva or blood may drool into pus basin by gravity.

The throat is sprayed four or five times with a 2 per cent nupercainepinephrin solution at intervals of a minute or so apart. No effort at rapidity is made; the technique is rapid enough as it is. The nupercain spray quickly quiets the gag reflex; next the tonsils and pillars are quickly swabbed with the same solution in an effort to locate any spot of mucosa where the gag reflex may be elicited.

A weaker nupercainepinephrin solution may be employed for peritonsillar injection. For a number of years 1 percent novocain, 1 per cent antipyrin, 1 per cent (1-1000) epinephrin has been employed for peritonsillar infiltration with such perfect results that I am loath to change this.

After the right tonsil is anesthetized, a minute or two delay is made before injection of the left.

Usually the right tonsil is picked up with the suction tube under direct inspection and footswitch control of air pump, the previously adjusted snare loop of No. 9 snare wire slipped over end of tonsil tube down in back of tonsil, tightened, the ratchet brought into play on Pierce-Mueller snare and the tonsil enucleated; virtually shelled out "in-capsule" from its fossa. This entire procedure of enucleation takes from 10 to 15 seconds as an average, more rapidly if desired. The enucleation is practically bloodless in the vast majority of cases. The tonsil is carefully examined for completeness of its capsule, then after about a minute the fossa is examined, using a tonsilloscopic tongue depressor (combined pillar retractor and tongue depressor) for perfect direct vision study of the entire fossa. Usually it is perfectly dry; if any oozing point is located, it is picked up with a Herbert tonsil hemostat and compressed for a few seconds. This is not required very often, and still less frequently is more than one oozing point detected. The heavy No. 9 snare wire plus the slow ratchet enucleation provides its own hemostasis as a rule, plus the fact that there is no sharp knife blade employed to plunge back of the capsule and open up bleeders. Ligature or suture is very, very rarely employed with proper suction technique.

When satisfied the first tonsil is perfectly enucleated and the fossa dry, the second tonsil is enucleated in the same way, the fossa carefully inspected as before, the Herbert hemostat employed if required, and the patient quickly put to bed, lying on his or her side with the mouth downward slightly, partly open, and the patient warned not to expectorate, cough or clear the throat, to simply permit any saliva to drool by gravity onto towel placed under mouth and changed as necessary. No pus basin is allowed around patient, to discourage efforts at expectoration.

Usually the entire operative transaction has taken less than two minutes; no pain, no fright, no bleeding, and a minimum of surgical shock, if any. The patient has been wide awake and observant throughout, but not frightened, and given just before operation the phenobarbital exerts a sedative effect but not time enough to develop its hypnotic action until after operation. Then, if left undisturbed, the average patient soon drifts off into a quiet sleep of two or three hours' duration, and awakens in good physical condition.

For postoperative pain a powder of morphin sulphate, gr. 1/32, in milk sugar, dropped on the tongue at three-hour intervals, is very effective. Nupercain spray or throat lozenges may be employed in conjunction, plus a liquid or semiliquid diet for the first two or three days. As a rule convalescence is rapid and uneventful, and a nice, clean, perfectly functioning throat the end-result.

Secondary hemorrhage after suction tonsillectomy must be very, very rare to judge by my observations over the past eight or ten years.

Tonsillectomy, in my judgment, is a difficult operation; sometimes a very difficult operation; and difficulties and complications are bound to occur with any technique and with any operator, and it is best not to criticize the end-results of any tonsillectomy because an anatomically and a cosmetically perfect throat immediately after enucleation is by no means a guarantee of a perfect final result.

If, however, our goal is to make the operation safer and less tiresome for the patient and easier and more satisfying to the operator, on any and on all types of tonsils, with local or with general anesthesia, I must feel that perfect equipment, perfect technique suction tonsillectomy, as one enthusiastic operator wrote me, "has the old methods all beat to a standstill."

And yet no claim is made that it is *the only way* to remove tonsils, no claim made that it is necessarily *the best*, no claim made that it is *perfect*, no claim made that it is *bloodless*.

An operator satisfied with his Sluder on any and all types of tonsils should continue with it, and so should the dissection and the electrocoagulation operator. All of these techniques have their places in the sun.

In brief, the only claim made for suction tonsillectomy is that it is worth, that it is entitled to, careful observation and study by every operator who wishes to make tonsillectomy *safer and less tiresome for the patient and easier and more satisfying to the operator*. This it does beyond question.

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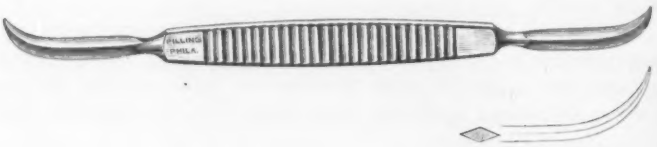
7 E. McMillan Street.

TECHNIQUE OF TONSILLECTOMY BY EVERSION.

Dr. H. H. Lott, Philadelphia.

If an apology is necessary for this explanation of a certain method of removal of tonsils, I can only say that I have been asked many times to explain the technique of tonsillectomy by eversion; and that is the first thing to firmly fix in the mind: the tonsils are *everted* and *not* dissected.

The tonsil is grasped with any ordinary tenaculum and pulled towards the median line. This will show the size and depth of the tonsil and where it is most adherent and will give information as to where and to what depth the evertor must be inserted. The so-called Lott Evertor (a flat, pointed instrument, curved at the ends in two different planes, on the edge and on the flat, as it is to be used merely as a hook or pry) is now inserted deeply into the body of the tonsil and the tonsil is everted or pulled through its own capsule, exactly as one would grasp the lining of a pocket and turn it inside out. This part of the operation causes exactly the same



position of the tonsil as any method where the tonsil is *pushed through*, as in Sluder or LaForce, etc., the only difference being that the work is done *in* the tonsil by pulling instead of by pushing against the pillar. When the tonsil has been everted, a Lewis Snare is placed about it. Traction is made towards the median line and the snare is tightened *slowly* to crush vessels and lessen the tendency to bleed (one-half to three-fourths minute for each tonsil being the usual time for tightening the snare). No. 9 wire is used, to lessen the tendency for cutting.

The advantages of this method are as follows: *1*. There is no unnecessary bruising of the surrounding tissue, nothing being touched except the tonsil itself. *2*. The period of healing is shortened because of lack of trauma. *3*. The ease of application under either general or

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local anesthesia. *4*. The short time necessary for its performance; two to five minutes for a complete T. and A. by either local or general anesthetic. *5*. The minimum amount of trouble with hemorrhage because there is no cutting or dissecting other than that by the wire snare.

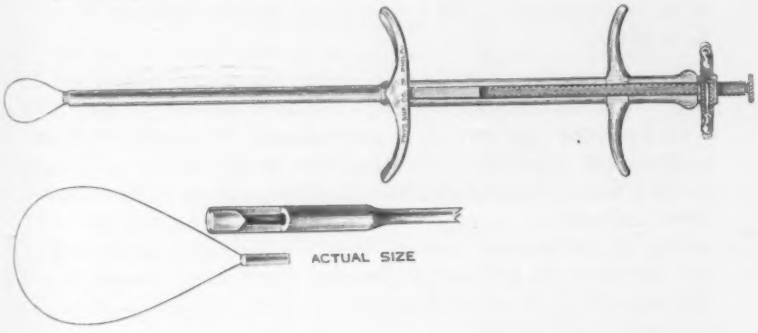
918 Medical Arts Building.

AN IMPROVED TONSIL SNARE.

DR. SETH A. BRUMM, Philadelphia.

In presenting an improvement on any surgical instrument it seems to me it is incumbent on the part of the designer to promote simplicity, durability, accessibility and thoroughness of function of performing the operation to which the instrument is applied.

Fig. 1 is a diagram showing my tonsil snare, which is almost self-explanatory.



A fine wire loop, namely, No. 3 or No. 4 wire, is used, which has almost the same cutting qualities as a knife.

Very easy for nurse to rethread as all bending of wire and seeking fenestra in stylets is obviated.

Impossible for wire to bind in cannula on reloading.

No spring, ratchets, etc., to keep clean.

1204 Medical Arts Building.

DECOMPRESSION OF THE FACIAL NERVE FOR FACIAL PARALYSIS FOLLOWING A MASTOID OPERATION. CASE REPORTS.

DR. J. MORRISSET SMITH, New York.

Case 1: Girl, age 10 years, with a history of a radical operation and complete facial paralysis on left side of the face one year and 10 months previous to this examination. Right ear normal, left ear filled with foul smelling discharge and granulations. Left side of face shows 40 per cent recovery of function. Left secondary radical mastoid operation and decompression of the tympanic and most of the mastoid segments of the nerve. Nerve found to be injured but not severed in the tympanic portion. Ear dry several months after operation, no loss of function following second operation, with an additional recovery of 30 per cent of function since the decompression.

Result: Ear dry, 70 per cent recovery of function, four-and-one-half years after secondary radical operation and practically a complete decompression of the tympanic and mastoid segments of the facial nerve.

DECOMPRESSION OF THE FACIAL NERVE FOR FACIAL PARALYSIS FOLLOWING A SIMPLE MASTOID OPERATION.

Case 2: Girl, age 6½ years, with a history of a complete facial paralysis, left side of face, following a left simple mastoid operation. the day before this examination. Right ear normal. The mastoid wound immediately reopened, nerve found to be injured but not severed in the mastoid segment from the horizontal, semicircular-canal almost to the stylomastoid foramen. Facial ridge lowered, mastoid segment of the nerve decompressed, drum and ossicles allowed to remain intact, flap cut.

Result: Facial paralysis completely cleared up four months after operation, 10 months have elapsed since the decompression, there is a slight mucoid discharge from the Eustachian tube, face normal.

123 East 53rd Street.

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*Read before the New York Academy of Medicine, Section of Otology, March 13, 1931.

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International Digest of Current Otolaryngology.

Editor:
Dr. Maxwell Fineberg, St. Louis.

Collaborators:
Prof. G. Bilancioni, Rome.
Mr. W. S. Daggett, London.
Priv. Doz. G. Kelemen, Budapest.
Dr. H. C. Rosenberger, Cleveland.

Lüscher, in the Zeit. f. H., N. u. O., 28:40:1930, describes reflex drum membrane movements and the function of the tensor tympani muscle in man. His observations were made with the Ohrmikroskop, which produces enlargement of about 20 times. With the aid of this instrument one is able to see movements of about 1/20 to 1/40 of a millimeter. It was ascertained that reflex drum membrane movements are rarely seen in humans. The movements seen with the aid of this microscope were very probably physiological in character and were not recognizable with the naked eye. Kelemen.

The Nov. 8, 1930, issue of the *Journal A. M. A.*, in its editorial column presents an interesting and complete resumé of agranulocytic angina. In view of the fact that otolaryngologists are frequently the first to see these cases, our specialty must acquaint itself with this condition in its early stages. The editorial stresses the fact that all cases of agranulocytic angina should be studied clinically and bacteriologically if anything is to be known about the etiology.

Muck, in the Zeit. f. H., N. u. O., 28:22:1930, described a nystagmus obtained by stimulation of the sympathetic fibres surrounding the blood vessels of the upper extremities. The stimulation may be either mechanical, thermic or electrical. The nystagmus is of a vasomotoric character and should be considered retrolabyrinthian. This phenomenon is of value in the differentiation between a labyrinthian and a retrolabyrinthian nystagmus, insomuch that stimulation of the radial artery should produce a retrolabyrinthian nystagmus. Kelemen.

Announcement is made that Professor George Portmann, of Bordeaux, France, will give a course of Ear, Nose and Throat Surgery at the University of Bordeaux, starting July 27, 1931. Professor Portmann limits his group to twelve and gives the course in the English language. Information may be had from Dr. Leon Felderman, 413 Mitten building, Philadelphia.

Vogel, in the Zeit. f. H., N. u. O., 28:30:1930, states that the galvanic nystagmus is of use in the localization of cranial injuries and in the diagnosis of Meniere's Syndrome Complex. He bases this on the fact that the labyrinthian dizziness may be traced to a difference in tone in the two vestibular apparati. This difference may be ascertained in most cases by the different responses to galvanic stimulation. A difference of one milliampere between the two sides to produce the same stimulation is considered pathological.

KELEMEN.

The Oto-Laryngological Clinic of the University of Naples, under the directorship of Professor Bruzzi, has collaborated with Dr. Alberto Pagano in the publication of a complete work on the Syndrome of Gradenigo. The author was an assistant of Gradenigo's and has gathered the material of his old teacher, and, as a tribute on the fifth anniversary of Gradenigo's death, Pagano and the Clinic published this volume.

The Syndrome of Gradenigo is discussed from the viewpoint of the originator and a complete reference on this subject is used to bring the subject-matter up to date.

The book is unquestionably a fitting tribute to the man who first described the Syndrome.

Wiethe, in the Zeit. f. H., N. u. O., 28:58:1930, discusses perforations of the esophagus, particularly those produced by esophagoscopy. He states that the esophagus tears in the long axis, even when damaged by penetrating instruments. The perforations are located mainly in the neighborhood of the cartilaginous rings. It is almost impossible to perforate the esophagus of a deeply anesthetized animal unless one deliberately sets out to do it. The same statement is true for the human cadaver. From this it must be concluded that the basic fundamentals for a perforation of the esophagus are a combination of the pressure of the instrument with a ring-forming muscular contracture.

KELEMEN.

The Faculty of Medicine, of Paris, announces that during June and July, 1931, a comprehensive course in Post-Graduate Medicine, including Otolaryngology, will be presented; the work will be directly under the sponsorship of the French government. Courses will be conducted in the English language and Clinics will be held in the great hospitals of Paris.

Detailed information may be secured by addressing Dr. Frank Smithies, 920 N. Michigan boulevard, Chicago, Ill.

Caesar Hirsch, in the Zeit. f. H., N. u. O., 28:140:1931, writes on the subject of malignant chordoma. He describes a case of a swelling of the right tonsil which extended to the pharyngeal wall. Excision for biopsy was diagnosed as carcinoma; later this swelling turned out to be a typical chordoma. Other writers have previously shown what a close similarity there is between the two conditions. Hirsch states that chordoma of the tonsil has not as yet been described, and he also states that never has a chordoma been found to develop so far away from its original embryological position.

KELEMEN.

F. T. Hill, of Waterville, Me., in the June, 1930, Annals of O., R. and L., writes on the importance of the history in the diagnosis of brain abscess. He reports several cases with unfavorable results in which operation was performed and later additional data was obtained which might have helped to form a more comprehensive diagnosis of the pathology present. Hill stresses the importance of the clinical history and considers his time well spent in questioning the relatives, family physician and other medical attendants. In his referred cases, Hill sends the previous doctor a questionaire asking him to check any of the enumerated signs of symptoms which he may have noted in the case, giving their time of onset. This seems to have given some measure of success in Dr. Hill's cases.

Muck, in the Zeit. f. H., N. u. O., 28:98:1930, discusses a method of distinguishing jugular thrombosis from an occluded sinus. The differentiation is based on the fact (especially in children) that when one jugular vein is compressed there is a peculiar noise heard over the other jugular, or an already existing bruit is increased. When this is done in a case of acute purulent otitis media and the increase is noted, then it points to an obliterated sinus.

KELEMEN.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Meeting of Jan. 9, 1931.

Anatomy and Pathology of Petrositis. Dr. Jos. G. Druss.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

The Symptomatology and Diagnosis of Suppurations of the Petrous Pyramid. Dr. Samuel J. Kopetzky.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Surgical Therapy for the Relief of Suppurations of the Petrous Pyramid. Dr. Ralph Almour.

Dr. Almour first presented a historical sketch of the various surgical measures that have previously been advocated for the relief of this condition. He described the methods advocated by Streit, Mayer, Eagleton and others. He then described his mode of approach for drainage of the petrous tip, which in brief can be summarized as follows:

A radical mastoidectomy is first performed and the root of the zygoma and the anterior canal wall are thinned down to an extent sufficient to bring into view the mouth of the Eustachian tube. The tensor tympani muscle and the cochleariform process are then removed. (Dr. Almour here exhibited anatomical specimens which showed the relationship existing between the basal cochlear turn and the carotid canal and between the apex of the cochlea and the Eustachian tube. Directly under the roof of the pyramid portion of the petrosa the basal cochlear turn is separated from the carotid canal for a minimum distance —14 specimens—of 6 m.m.)

With the use of a 1 or 1½-m.m. burr directed at an angle of approximately 20-25° to the axis of the external auditory canal, an opening is made just in back of the Eustachian orifice, directly under the tympanic roof. The burr then passes into a pyramidal space, the sides of which are formed by the carotid canal in front, the three turns of the cochlea behind and the Eustachian tube externally. The apex of the pyramid is below and the base above. Through this space access to the petrous tip is possible for drainage of a suppurative focus within it.

Dr. Almour discussed the indications and contraindications for this procedure, stating that only injury to the carotid artery is to be feared. This can always be avoided if it is remembered that the uppermost portion of the carotid canal is below the level of the roof of the Eustachian orifice. Therefore, if the petrous tip is entered at the level described by him and in the area described, the carotid canal is avoided.

He stated that this procedure should never be used in cases wherein the abscess within the petrous tip has ruptured subdurally and formed an extradural abscess. In such an event, the extradural abscess should be drained after the method described by Dr. Clarence Smith at this meeting.

DISCUSSION.

DR. WELLS P. EAGLETON: I wish to thank and to congratulate the different gentlemen on the very fine work that has been presented tonight. Dr. Druss' slides were most interesting; perhaps he will let me take them home and study them some time.

Dr. Kopetzky has reported a number of cases of a specific type with a large percentage of recovery. Dr. Almour has devised an operation that, based on anatomical grounds alone, the Section should be proud of, for it is a highly technical one and he has found a way through the middle ear into a hitherto inaccessible part except by way of the middle and posterior fossae.

Now, what have I learned by their papers? It has been stated that there is extensive pneumatization in all these cells. I do not think this is so. But I have learned that there are two types of cases, one like Dr. Smith's case (osteomyelitis) and one like those reported by Dr. Kopetzky (disease of pneumatic cells). I have thrown them all into one group; I thought that one was the result of the other.

I have long known that labyrinthitis coming from behind is different from one coming from in front; but I never had its clinical course impressed upon me as in Dr. Kopetzky's paper.

Then about the profuse otorrhea; the main reason that many of them are profuse and continue so is because of the involvement of the petrous apex. That has never occurred to me.

Now, after all has been said on the supposition that the disease is in the pneumatic cells in the apex; that pneumatization has extended in and around the labyrinth through the peritubal spaces into the petrous apex, and that the petrous apex usually is a pneumatic space is contrary to my experience. The petrous apex may be pneumatic, but it generally is not; the petrous apex is generally a bone producing marrow and that is why it is different from the rest of the temporal bones. We must throw out of our minds suppuration in the apex that is based on what occurs in mastoid disease. In the body we have flat bones and long bones, but the temporal bone is a capsular bone and it has two parts that are entirely different from any other bone. The mastoid bone is a pneumatic space bone. When the child is first born the mastoid is filled with fat, but as the mastoid develops it grows by pneumatization; this growth entirely by pneumatization occurs nowhere else in the body—not in the ethmoids nor sphenoids. It is a different developmental process. On the other hand, the petrous apex has the characteristic of the metaphysis end of the epiphysis of a long bone. It is the growing end of a long bone. It is as if a femur had been cut off here and a growing end of the femur filled with bone marrow had been placed transversely.

In the cases spoken of tonight, all have been treated as if pneumatization coiled around the labyrinth, but the anatomical fact is that few of the cases of petrous apex suppuration have pneumatic spaces in the apex; they have bone marrow spaces in the apex; and so we have in the apex a type of osteomyelitis that you have nowhere else in the temporal bones. The osteomyelitis of the squama and mastoid (Heiberman) all occur before the age of 10 years. Why? Because the mastoid process is not a blood producing bone. Now, if you have a pneumatization of the apex of the petrous, then you normally should have a cavity lined with epithelium. And how is it, if the apex is the same as a mastoid, that just letting out a few drops of pus cures the case? We know one cannot open the mastoid and let out a few drops and it will continue to suppurate for a very long time. But the fact is that here in the petrous apex a small drill hole is made and a few drops of pus evacuated, and there is recovery. Why? Because in the petrous apex there is blood producing marrow and it has a distinct phagocytic property, it itself controlling suppuration; so if you let out the pus that may be there, the case will do well. We know that the orthopedic surgeon in osteomyelitis of children does not remove the whole area, but makes an incision through the periosteum, and the osteomyelitis takes care of itself.

I am glad to have it called to my attention that there are two types of cases, one which has to deal with disease of a pneumatized apex, and another in which we are dealing with an osteomyelitis.

Now, there is one point in a paper on "Unlocking of the Petrous Pyramid," that I read before the Triological Society, January, 1930, and which was submitted to the Archives of Oto-Laryngology some time this last summer and accepted for publication. It is a long paper, dealing with the pathology of osteomyelitis of the apex and report of cases. One case, I believe, was the first case in which an osteomyelitis of the apex had developed a meningitis with recovery of the patient. The principle of the operation is this: The external bony removal is very similar but much more extensive than in Dr. Almour's operation, but he goes into the apex from the middle ear and I enter from the middle fossa. In the paper I have described how, if the developed

part of the squama, the tympanic ring and the glenoid tuberostity are removed and the anterior buttress, the roof of the middle ear removed and the floor of the middle fossa lowered—the labyrinth and tube are plainly seen by direct inspection. You look down directly at the labyrinth. The idea is to convert an adult bone into exactly what is present at the time of birth.

If the labyrinth is still functioning the dura is now elevated and the bone of the apex perforated.

If the labyrinth is dead the apex is entered through by eviscuation of the bony and membranous part of the cochlear. The opening into the apex by the latter method is large enough to allow of complete curetting of the whole cavity—as well as drainage—but this is generally unnecessary as perforation of the bone alone is all that is generally necessary. So there are several types of cases, and no one operation will solve them all. Again, I congratulate these gentlemen on having made a very valuable contribution to the whole subject.

Dr. M. A. Goldstein: It is a pleasure to extend greetings of fellowship from the Middle West to the members of the Otological Section of the New York Academy. The presentation of this program has been an inspiration to me. I feel that the men who have contributed to this symposium on Anatomy, Pathology and Surgical Draining of the Petrosal Tip have made a new chapter in otological history tonight. Our friend and colleague, Dr. Eagleton, instigated this chapter by his medical philosophy and the characteristic title of his written message, "Seeing Daylight Through the Petrosal Pyramid."

The surgery of the petrosal pyramid has always been regarded as a formidable and serious one and the prognosis and results of such surgery until now have not been hopeful.

It is a significant fact that of the nine cases of petrosal pyramid suppuration reported tonight, so large a proportion have recovered as a result of the described new technique. To me, the most significant fact in the problems of our surgical duties is not *is our philosophy and operative technique correct,* but, *what is the end-result for the patient?*

I wish to congratulate the participants in this symposium for the three excellent presentations of anatomical and pathological description, report of clinical experiences, and the operative technique as developed by Dr. Almour. We are again proud of American otology in the contribution of this distinctly valuable piece of research.

I thank you for the privilege of hearing this symposium and speaking to the Section.

Dr. Edward L. Berger: May I relate an interesting case under my supervision at present, which will bring out a few of the points that have been elicited tonight? It is that of a boy, age 17 years, who was well until Nov. 6, when he developed an otitis media with spontaneous rupture on the sixth day. On the eighth day he developed supraorbital and deep orbital pain. He was operated upon on Nov. 22 for coalescent mastoid. There was no exposure of the dura or tegmen of middle fossa. A granuloma was found on emissary vein where it joins the lateral sinus. The orbital pain was relieved for four days after the operation, and then it recurred. On the fifth or sixth day postoperatively a lumbar puncture was done. We found 1300 cells, 52 per cent polys., and 48 per cent lymphocytes. The pain over the right eye and in the orbit persisted, nocturnal in character, with low grade temperature, 99° to 100.5°, and we decided to explore. We explored the petrous pyramid from behind, and exposed an area of dura in middle fossa, the size of a quarter, and I found nothing. The mastoid wound was healing nicely; there was very little discharge from the middle ear or antrum and there was healthy granulation tissue. We then did a second lumbar puncture four days after the second exposure, and we found about the same as the first count, only 48 per cent polys., instead of 52 per cent. The headaches were relieved after the second lumbar puncture and he was sent home. A few days later the temperature was almost normal. One week after his return home he had a similar attack of headache and orbital pain, and about five weeks after the first operation, when the wound was almost healed and the middle ear was almost dry, he had a terrific gush of pus from the middle ear and into the almost healed mastoid wound. This was on Dec. 25, 1930; a third lumbar puncture showed a normal fluid on this day. With the discharge of the pus, the headache was

entirely relieved and he has had none since. On Dec. 30, a day before the new year, he developed an external rectus paralysis. He had no headache, his temperature was normal, and he has a dry ear now and an almost healed wound. I revised the granulation tissue with the recurrent infection.

He had only a simple mastoid operation, and it seemed to me that the pus was coming from the superior anterior area of the attic. Probably peritubal cell infection.

I would like to ask Dr. Kopetzky whether the boy had a petrositis and his probable future course, etc. Is he in the quiescent stage? I am treating him expectantly and hope he will be well in a short time. His external rectus paralysis is subsiding, he has trouble only at certain times of the day, and is feeling very well. Temperature perfectly flat. No pain in or about his right eye.

DR. MAYBAUM: The subject of petrositis is best approached and studied from a pathological and histological standpoint. Dr. Druss has referred to two types of petrositis; one in which suppuration had extended to the tip, the other in which there was a perforation on the posterior aspect at a considerable distance from the tip. The clinical picture would surely differ in these cases. The degree of inflammatory change in the petrous bone does not coincide necessarily with the clinical course.

Most of the facts presented by Dr. Kopetzky are in agreement with those we hold. I do not believe that the clinical entity to which he refers definitely establishes the presence of a suppurative focus in the petrous pyramid in every instance. One may often go astray in deciding upon operative indications from the group of symptoms to which he refers. One experience has been that sixth nerve involvement is commonly associated with involvement of Gasserian ganglion. We have not seen seventh nerve involvement with vestibular symptoms in our cases.

In presenting his two cases, Dr. Smith gave a practical approach to the surgical problem connected with this condition. He reoperated and found a lead into the petrous pyramid through one of the areas in the mastoid referred to by Dr. Friesner, *i. e.*, the inner aditus wall. In addition the cells in the solid angle and the peribulbar region should be thoroughly exenterated. Only if the clinical course does not improve after this procedure, should a radical mastoid be done and any further leads into the petrous bone be investigated. The peritubal cells which are curetted in doing the last steps of a radical mastoidectomy should receive more than the usual attention.

I feel that the operation proposed by Dr. Kopetzky and Dr. Almour is a clever one, but not unattended because of the anatomical relationships with considerable danger. To my mind, a clearer differentiation of the clinical types of cases presenting petrous pyramid involvement must be evolved, before definite indications for surgical interference can be advised.

DR. J. M. POLISAR: I have a case under observation which Dr. Kopetzky is familiar with. The patient is a girl, age 19 years, who came to me three weeks ago complaining of intense pain in her left ear. She had a bulging drum, with marked tenderness over the mastoid tip and antrum, and a temperature of 102° F. Paracentesis of the left ear was performed.

She gave a history, starting four years ago, of having had a feeling of numbness in the lower half of the body, which lasted for a few months. About a year-and-a-half ago she suddenly put on weight (about 50 pounds within three months). Seven months ago she had left facial paralysis, which subsided within three weeks, followed by diplopia and ptosis of the left upper eyelid, which also subsided within two or three weeks. For the past seven months she has had the sensation of falling to the left. She could not walk alone, and would bump into the person with whom she would be walking (ataxia). For the past six weeks she has been complaining of dizziness and vomiting, usually projectile. She had a horizontal and rotatory nystagmus occasionally to the right, but mostly to the left; also a vertical nystagmus on looking upward. A week after the ear was opened her temperature was still 102° with profuse purulent discharge, and tenderness over the mastoid more marked. X-ray showed obstruction of the left mastoid cells. I was rather disappointed, when operating on the mastoid, I found only a few drops of pus in the antrum, some granulations and necrosis around the tegmen antri and sinus tegmen angle.

There was no exposure of dura or sinus. However, after the operation she stopped vomiting for four days, for the first time in six weeks, and her temperature dropped to normal. On the fourth day her temperature began to climb again, with the nystagmus more intense but not of the labyrinthine type, as the quick component was directed toward the diseased side. She also complained of pain in the left eye. Dr. Kopetzky saw her and thought it might be a case of petrositis; he also suspected a tumor or abscess of the cerebellum. She continued to have temperature for ten days. For the past four days her temperature has been normal and the nystagmus, vomiting and dizziness have disappeared. The patient now feels quite well. I wonder if this is not the stage of quiescence, the third stage which Dr. Kopetzky mentioned?

DR. J. G. DRUSS: I wish to express my thanks to the gentlemen who in their discussion have commented so favorably on my paper. There is one point I would like to make clear, and that is that while the infection may spread through the contents of the petrous pyramid and involve the apex, it frequently breaks through the bony cortex in its course to involve the dura along which it spreads to reach the apex. The ultimate clinical picture may be the same, but the underlying pathological condition in each case cannot be diagnosed before operation. At operation the route of extension may be noted.

DR. S. J. KOPETZKY, in closing the discussion, said he did not like to leave the discussion "up in the air" and that, therefore, despite the lateness of the hour, he would take up the points raised by the various men and would endeavor to clarify them, because some confusion was apparent, which it was necessary to dispel.

Dr. Friesner advocated waiting until signs of intracranial involvement were more evident. This is precisely the crux of the situation: it is exactly what we do *not* do. We contend that suppuration of the petrous pyramid is a clinical entity which we have been able to identify. Its symptomatology is manifest, while the pathology is limited to the contents of the petrosal pyramid. It has characteristic, significant signs, which I have described in my paper, and we have been able to combat it successfully by using the surgical technique which Dr. Almour has described.

Under such circumstances, Dr. Kopetzky asked, what reason is there to await intracranial infection before intervening surgically? Once a meningeal infection is established, every otologist is aware of the gravity of the situation and of the lack of success procurable by the best treatment.

Dr. Kopetzky said that he was referring exclusively to suppuration of the petrosal pyramid in well pneumatized tips. In the others, where the petrosal pyramid is diploeic or nonpneumatized, we usually get an osteomyelitis. No cases of this type were included in the reports made by Dr. Kopetzky or Dr. Almour this evening. Such cases form an entirely different clinical group, which requires a totally different method of surgical attack. Dr. Kopetzky agreed with Dr. Friesner that retro-orbital pain is sometimes present in sinus thrombosis; but retro-orbital pain, accompanied by a low grade sepsis and by Roentgenological findings of a broken down petrous pyramid, after prior X-rays had shown a well pneumatized mastoid process and an undestroyed, pneumatized petrosal pyramid, constitutes a different picture from, and has a different significance than, retro-orbital pain, accompanied by a typical septic temperature, a Roentgenographically normal petrosal pyramid and other signs of sinus thrombosis. The diagnosis can be made whether the ear is dry or discharging at the time of examination.

Replying to Dr. Eagleton, Dr. Kopetzky said that osteomyelitis of the temporal bone constitutes an entirely different type of case from that which formed the basis of his paper. In osteomyelitic infections we deal, not with pneumatized, but with diploeic, petrosal pyramids. I attempted to account for the reappearance of otorrhea, or for its persistence, after simple mastoidectomy in cases of acute coalescent mastoiditis, where the operation had been properly performed. I made this point clear at the recent meeting of the Eastern Section of the American Rhinological, Laryngological and Otologcal Society at Atlantic City. The recognition of the lesion we are describing accounts for the finding of a persistent or recurrent otorrhea in these cases. It does not imply that it occurs in diploeic petrosal pyramids or in osteomyelitic lesions which occur in diploeic bone. The proportion of diploeic or pneumatized

petrosal pyramids may be as Dr. Eagleton asserts; but tonight I am concerned only with the lesions which occur in pneumatized petrosal pyramids.

Regarding the time to operate, Dr. Kopetzky again stressed the fact that surgery is indicated while the lesion is still confined within the bony boundaries of the petrosal pyramid. When the lesion is already in the endocranium, we have another situation. The cases reported by Dr. Smith earlier in the evening evidenced this. Dr. Kopetzky stated that he did not claim that a cure cannot be obtained by operating through the endocranial cavity; but it is as unnecessary to open into the endrocranial cavity when eradicating a lesion confined to the petrosal pyramid as it is to open the middle, or the posterior, cranial fossa when doing a mastoidectomy for a coalescent mastoiditis in which the disease is limited to the mastoid process. Approaching the lesion endocranially does not lessen, but increases, the surgical trauma and risk.

Dr. Kopetzky said that Dr. Maybaum confused the discussion by introducing the element known as the Gradenigo syndrome. Dr. Kopetzky again stressed the fact that none of his cases of suppuration of the petrosal pyramid presented an involvement of the sixth nerve; and he drew Dr. Maybaum's attention to the fact that recent otologic literature, both here and abroad, had discarded the concept that the Gradenigo triad is significant of any one lesion or characteristic of a particular clinical entity. In the course of suppurations of the petrous pyramid, the sixth nerve is occasionally involved in the third or fourth period of the disease. Dr. Kopetzky stated that he had probably seen as many sixth nerve involvements as Dr. Maybaum, the majority of whom had recovered without resort to surgery. They were, however, not cases of suppuration of the petrosal pyramid; and undoubtedly neither were Dr. Maybaum's cases of this group.

Dr. Kopetzky reiterated that the surgical procedure devised by Dr. Almour was feasible. It was practicable and it accomplished what it set out to do: *viz.*, furnish free and adequate drainage of a suppuration in the petrosal pyramid before the suppuration ruptured into the intracranial cavity. To perform the operation, an exact knowledge of the surgical anatomy of the parts involved was necessary; but the operation in itself, when properly performed, entailed no untoward risks.

In conclusion, Dr. Kopetzky thanked all who had taken part in the discussion and expressed the hope that further work would substantiate and clarify the views expressed.

Dr. Ralph Almour: Dr. Maybaum and Dr. Friesner have cautioned us to look for other sources from which pus might come before we enter the petrous tip. I took that for granted in the presentation of my paper. Before the petrous tip is entered, a search is made of the peribulbar area, the area around the petrous base, etc.; then when the simple cavity is converted into a radical one, all the areas which might be involved in the antrum and epitympanic space are carefully inspected, and if necrosis is found these areas are probed. All these cases should be inspected very carefully to determine the area of invasion, and if that be determined, the tip should be entered if the symptoms Dr. Kopetzky has outlined are present. Dr. Maybaum calls the operation I have devised "dangerous." It is not more dangerous than the simple mastoid in the hands of a tyro; anyone who does not know the anatomy of the mastoid process should not be permitted to do a mastoid operation; and similarly, in order to perform this procedure, a clear and accurate knowledge of the anatomical structures present and to be avoided must be known. If one is familiar with these, there is no danger. The entrance to the carotid is the one danger that presents itself, but if the burr is placed at the high level described, before the superior wall of the carotid becomes membranous, one cannot injure the carotid artery. If the carotid be exposed and bathed in pus, the canal should be drained. Again, this operative entrance into the petrous tip is not advised in any case that has ruptured subdurally. If a subdural abscess is present it is a complication; and since it is such, it should be treated as any such complication; namely, by approaching the area involved and draining it directly, as Dr. Smith has done.

I wish to thank Dr. Goldstein, Dr. Eagleton and the others for the very kind treatment I have received from them.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Meeting of Feb. 13, 1931.

DR. ARTHUR B. DUEL: I fancy that the reason for the invitation to me to open this meeting by a few introductory remarks is due to the artistic nature of the officers. They would no more think of plunging in *media res* with this show than Gatti-Casazza would think of putting on Pagliacci at the Metropolitan Opera without the overture.

Since brevity is the soul of wit and tediousness the limbs and outward flourishes, I will be brief. I shall take for my text then, from the Gospel of St. Matthew, the seventh chapter and twentieth verse: "Wherefore, by their fruits ye shall know them;" and having stated the text, my theme is developed and my sermon ended.

There is another quotation: "Good wine needs not the bush," but that carries with it a sense of boastfulness which I would avoid. We had better bring on the actors and let them speak for themselves.

I must grasp this opportunity, however, to congratulate the officers of this Section, as well as those of the Nose and Throat Section, on the increasing number of sessions at which clinical presentations are the feature of the evening. After all, it is only occasionally that a prepared paper is of great interest. The interesting one is that which probably requires careful reading later on. As a result, as one grows older, he is liable to find excuses more readily for nonattendance. In meetings like this, where cases are shown and discussed freely, one is bound at least to have more interest and, I believe, usually derives more benefit.

Mastoidectomy Complicating Cervical Pott's Disease. Dr. Arthur E. Warren.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

End-Results in 20 Cases of Primary Skin Grafts in Mastoidectomies. Dr. Daniel S. Cunning.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

A Case of Jugular Thrombophlebitis Extending Into the Innominate. Dr. Wright MacMillan.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Case of Temporosphenoidal Lobe Abscess Complicating Chronic Otitis Media Purulenta. Dr. H. M. Sheer.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Malignant Tumors of the Ear. Dr. G. Allen Robinson.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Report of a Case of Acute Diffuse Labyrinthitis. Dr. Lester Mead Hubby.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Report of a Case of Secondary Simple Mastoidectomy With Choked Discs and Severe Cerebral Symptoms. Dr. H. M. Sheer.

(Appears in full in this issue of THE LARYNGOSCOPE.*)*

Case of Simple Mastoidectomy Followed by Cerebellar Abscess; Operation; Recovery. Dr. H. Clifton Luke.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Deformity of Ears and Nose from Lupus Erythematosis Benefited by Intracutaneous T. B. Inoculations. Report of a Case. Dr. Edmund P. Fowler.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

A Syndrome of Avellis Variation. Case Report. Dr. Edmund P. Fowler.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. CHARLES M. WILLIAMS: This case is an interesting one from many points of view. First, the diagnosis: I agree with the diagnosis of lupus erythematosus, and yet it presents some features which make one think very strongly of lupus vulgaris—the extent of the disease and more especially the destruction of tissue; the nose and two ears are practically gone. This is not common in lupus erythematosus, which has a thin, pliable scar with no constriction and no deformity; and yet we have these unusual cases occasionally where lupus erythematosus goes on to this destruction. It is interesting in this connection to mention that there is a form of lupus vulgaris which resembles lupus erythematosus very closely, so it is very interesting that tuberculin has been effective in a case of lupus erythematosus, which has features suggestive of lupus vulgaris.

The etiology, as Dr. Fox said, is doubtful—some disclaiming any connection with tuberculosis; but the majority of observers claim that some of the cases at least are tuberculous. I think that qualifying *some* is the essential point. Lupus erythematosus is not a thing with a single etiology. The toxin may be tuberculous or from some other organism, or it may be a toxin produced by the body and not from a germ. But there are cases in different sets of statistics which seem to be definitely tuberculous. This is determined by the examination of tissue and, as in this case, by the successful result of tuberculin treatment. As only a fraction of the cases are caused by tuberculosis, it is natural that statistics should vary. If the observer is in a community where tuberculosis is common, his cases may be largely tuberculous; whereas in another region this factor might be negligible.

A case was quoted by Dr. Fowler of a patient in whom a flare up was caused by a single injection, which produced marked symptoms: Why that sometimes occurs, at other times not, is a problem for the anaphylactic experts and beyond me, but such results are observed.

Dr. Fowler is to be congratulated on the results of his treatment in this case. The man is definitely improving, and I would continue with that treatment. I do not see any reason for changing to something which is usually considered better. If the improvement ceases, I would change to gold and sodium thiosulphate—but not before. We are now trying at the Skin and Cancer Hospital the injection of gold and sodium thiosulphate, with novocain, directly into the lesion itself, and some cases are improving which have been resistant to the intravenous treatment.

DR. HOWARD FOX: I appreciate the kindness in being asked to discuss this interesting case of Dr. Fowler and congratulate him on the results of treatment. Lupus erythematosus is a fairly common affection of adults and in the ordinary fixed type appears most often on the nose and cheeks, but may also affect all parts of the ear, the mastoid region and the scalp. It consists of well defined, reddish, dry patches, which do not itch and which are more or less covered with finely adherent scales. It is an exceedingly chronic disease, eventually causing atrophic scars and until the recent advent of gold therapy was a most difficult one to treat.

The etiology of lupus erythematosus is still a matter of dispute. Some think that all cases are related to tuberculosis and class the disease among the so-called tuberculids, due either to toxins or to dead or attenuated bacilli. In a series of 250 cases collected by Roth there was evidence of either local or general tuberculosis in 180 of them. MacLeod, of London, estimates that one-third of the cases are associated with tuberculosis, the others being due to focal infection with other organisms, often derived from the teeth. Tubercle bacilli in rare instances have been found in lupus erythematosus and a few positive animal inoculations are on record. It is interesting to note, however, that histologically there is not the slightest suggestion of any changes characteristic of tuberculosis.

Opinions seem to differ about the value of tuberculin therapy in this disease. Oro, in a series of 51 cases, obtained cures in 17, *e. g.*, one-third of his cases. Korlisch cured eight out of 10 of his cases, while Sieben had a successful result in one case of 20 years' duration. There was no recurrence at the end of 18 months.

On the other hand, Cannon and Ornstein in a series of 15 cases obtained apparent cures in only one-fifth of the cases. MacKee in six cases saw no beneficial result. Hoffman stated that after a single administration of tuberculin the eruption became generalized over the entire body. Others have reported severe reactions, and even deaths.

I have had no personal experience with tuberculin in lupus erythematosus but might add that the most favorable results are apparently obtained at the present time by various gold compounds, the favorite preparation in this country being the gold and sodium thiosulphate.

DR. ARTHUR B. DUEL: I am somewhat overwhelmed by the wealth of material presented tonight. I remember when I first entered the realm of otology, some 30 years ago, the otologist was facetiously described as "the specialist who treats incurable cases." I well remember in those days a meeting at the Section of Otology of the Academy when I was enthralled with the report by Dr. Whiting of a case of septic sinus thrombosis which had been operated by Dr. Robert Abbe, a prominent general surgeon. A brilliant report of excision of the jugular vein and blocking of the lateral sinus which had resulted in recovery of the patient. Tonight we have listened to the reports of a series of cases which at that period of the specialty would have thrilled the otological world. This only emphasizes the fact that the otologist has come into his own. The otological surgeon is now regarded as a real entity, an important specialist in the surgical profession, who himself does all the surgery of otitic origin. The hour is too late to attempt to enter into a discussion of many interesting features of these reported cases. Indeed, the cases speak for themselves.

DR. JOHN R. PAGE: I feel as Dr. Duel does, that there has been such a wealth of material presented tonight there is no time left for its discussion. Beginning with Dr. Warren's paper, there are many points of interest to discuss, but the hour is too late. Dr. Duel referred to Dr. Whiting. I would like to say that I don't know of any method of exploring the brain that is less harmful and more accurate than his of making separate parallel explorations through small dural incisions. Following this method, there were numerous explorations made in the temporosphenoidal lobe and cerebellum in Dr. Sheer's case, and no harm resulted. If some other method had been tried, the result might not have been so fortunate.

There is hardly time to more than mention the case I was to report tonight, of a woman, age 28 years, who had a cerebellar abscess in the opposite side of the cerebellum from the mastoid, on which she had had a sinus and jugular operation on Sept. 11. There was some indication of a pathway of infection through the inner wall of the lateral sinus to the cerebellum on the right side a few days after the sinus and jugular operation. On Sept. 18 there were signs of developing brain abscess, which became more marked two weeks later and then entirely subsided, and the patient left the hospital. Two weeks later she returned to the hospital, with definite signs of an abscess in the left side of her cerebellum behind a normal middle ear and mastoid and away from the infected side, on which the sinus operation had been performed. An operation was hurriedly performed after the patient had stopped breathing and was undergoing artificial respiration, and the abscess was discovered in the left side of the cerebellum. There was a spectacular return of respiration as soon as the abscess was tapped through a small opening hurriedly made. At two subsequent operations, the endothermic needle was used with some degree of success in making a better opening to the abscess and in removing a large hernia that resulted after the second operation. The patient is alive to date with a small recurrent hernia, normal temperature and fair mentality, but with little evidence of compensation as yet for what seems to be the complete loss of the left side of her cerebellum.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, March 3, 1931.

Presentation of an Instrument for Teaching Purposes, Demonstrating the Normal Flow of Lymph Currents in the Semicircular Canals During Rotation Test. Dr. M. Valentine Miller.

(Appears in full in this issue of THE LARYNGOSCOPE.*)*

Case Presentation: Lymph Hemangioma of the Nose, Tongue and Soft Palate. Dr. M. A. Zacks.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. STRAUSS: This rather unusual case has been very ably presented by Dr. Zacks. In a rather large experience with nevi—conservatively, I should say that I have removed 400 to 500—I can recall none involving the mouth and tongue, with the exception of a few situated on the lips and adjacent structures. Whereas nevi are very common, those involving the tongue must be rare, for it is certain that if they existed they could not be easily overlooked, for practically everybody's mouth is at one time or another inspected by the dentist or doctor. In the thousands of mouths that you laryngologists have examined, how many nevi can you recall seeing on the tongue? In Butlin's book on diseases of the tongue very little space is given to nevi.

But then the rarity of the condition is by no means the only unusual feature in this case. First, in practically all the reported cases the lesion occurs on the anterior third and on the side of the tongue. Here it is on the posterior third and in the middle. Secondly, multiple nevi are not so common. And thirdly, there are two different types of lesions—lymph and hemangioma—so close together that they appear as one single lesion.

As was pointed out before, nevi are exceedingly common, occuring in some form or other in practically everyone, but their cause is unknown. While the emotional disturbance theory in the mother during pregnancy—shock, fright, illness, accident—has no scientific basis, this theory should not be entirely ignored for there are many cases cited by good authorities in support of this view. Heredity plays an important part and certain types are handed down for several generations. That they are connected with imperfect development is evident from the frequency with which they are associated with physical defects such as harelip, cleft palate, arrest in mental development, and from their occurrence in portions where developmental errors are liable to be found—midline of body, embryonic clefts, nasofacial sulcus. In the case presented tonight, the midline is involved along with a congenital eye lesion of some type. Virchow suggested that nevi had an anatomical basis and were dependent for their situations on embryonic fissures—fissural angiomata. This only partly explains, because they occur on any part of the body and not only at embryonic clefts. Unna ascribed the condition to intrauterine pressure or to pressure at delivery on account of their frequency on the forehead and occiput, but this does not explain them all. I feel quite certain that the occurrence in this case of all these lesions in one region cannot be explained by the anatomy of the venous supply to the parts unless one were to assume some unusual blockage of pressure to the internal jugulars, and I believe there would be more serious symptoms if that were the case.

What about the removal of nevi and the methods to be employed. My feelings have always been that they should all be removed and removed very early in life. As Dr. Zacks pointed out, there is not much danger of malignancy, but those on the tongue and in other situations which are subject to continuous irritations are liable to infection and hemorrhage. Also at times they tend to enlarge and, of course, when they occur on the tongue there is

the danger of getting obstructive symptoms. End-results are much better when they are removed early, especially from a cosmetic standpoint, and that is certainly to be taken into consideration.

Dr. Zacks has mentioned the different methods that may be employed, but undoubtedly radium is the one of choice in this case. The ease with which it is employed—absence of pain—makes it the ideal, especially in children, and the end-results when properly applied are excellent. Either radium or radon in the form of an applicator can be used on all the lesions in this case. The lesion on the tongue may not yield to radium as growths arising from the lymph structures do not yield as readily to radium as those arising from the blood vessels. In closing, I may say that great care must be exercised in removing nevi with radium so that no permanent disfiguring damage will result from the radium itself.

Mastoiditis in a Case of Clinically Negative Diabetes. Dr. Geo. W. Mackenzie.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. SHEMELEY: It was my pleasure to assist Dr. Mackenzie in the care of this patient. He has covered the case very thoroughly and there remains but little to discuss. However, I might mention the fact that here was a surgeon whose brother was also a physician who had implicit confidence not only in Dr. Mackenzie, but also in the various consultants called in. I am quite sure that if Dr. Mackenzie had had as a patient an ordinary individual and had not been hampered by many relatives and many consultants he would not have had quite so difficult a time. The pathologist on the case assumed the attitude that all the treatment the patient was receiving was wrong. We were always taught in the army that it was right to criticize anything if you had somehing better to offer, but this man had nothing better to offer. His attitude became so peculiar that I was led to ask him if he did not believe blood transfusion would do any good and if he did not believe operation would do any good, just what he had to suggest to take their place. He had nothing to suggest. We used these methods and the patient recovered.

This brings up the question of Normet's solution. This patient received several injections of this solution. Dr. H. L. Northrop, of Philadelphia, has had much experience with it. I think he has stopped doing blood transfusions. He says there is no necessity to wait and you don't have to go to all the trouble of a transfusion. It is a preparation that was brought out by several French investigators. It consists of sodium citrate, calcium magnesium, ammonical iron and manganese completely dissolved in distilled water.

This case developed a facial paresis late and the recovery was complete. It is well to remember that those cases showing disturbance of the facial nerve some time following operation have a paresis and most all recover. Those in whom the disturbance of function of the facial nerve is immediate have a paralysis and do not recover without plastic nerve surgery.

DR. STOUT: If you have ever done general practice you have had occasion to see diabetes with negative findings. One of our professors said, "Never trust one examination." Both urine and blood may not show it at times, especially if the patient has been fasting.

DR. SHUSTER: The case presented by Dr. Mackenzie is very interesting and is a typical case of mastoid infection with lateral sinus thrombosis taking the usual course, with some complications, and is a record of work excellently done.

There is one thing, however, he has not made clear in my mind and that is the connection of diabetes with this particular case. First of all, his only contention was the clinical appearance of the wound. It is a question as to whether or not this clinical observation is sufficient to overcome the report of 111 m.g. blood sugar, and establish the fact that diabetes exists. With all of these manifestations during the course in this case of sinus thrombosis, *e. g.*, pyemia causing an abscess of the shoulder, it is possible that this sugar condition resulted from the pyemia, involving the pancreas.

There is one point I would like to mention about facial paralysis and that is the fact that it comes on immediately after operation does not necessarily mean it is permanent. I recently had a case that came out of ether with facial paralysis and recovered completely within two weeks.

DR. ERSNER: This most interesting paper by Dr. George W. Mackenzie lends itself to discussion from two angles. There is no doubt from his description of the case reported that it is a typical case of sinus thrombosis and I am unable to see his reason for clothing the subject in so mysterious a manner. I want to congratulate him on his clinical observation in recognizing diabetes from the postauricular tissues. There is no doubt that there was some slip-up in the blood sugar laboratory technique; otherwise, this diagnosis would have been made earlier in the disease.

In regard to the sugar problem, several years ago we had a number of patients who demanded consultants, irrespective of whether this was warranted or not. At that time we made a special study of patients who had prolonged postoperative temperature. Our routine study consisted of a basal metabolism and a blood sugar test. We found an increase of 20 to 30 points in the basal metabolism in some instances and a hyperglycemia in many of these cases. From your early blood sugar report, which was, if I remember correctly, 105, which is normal, we can only deduce that the first and second reports were incorrect. It is also reasonable to assume that the hyperglycemia found late in the disease may be attributed to infection and the toxic factors or it was due to secondary foci involving the pancreas. When there is persistency of high temperature between 105° and 106° as in this case, there may be an increase in the blood sugar which may be transitory; on the other hand, if it is a secondary foci, it may involve the pancreas as well as the shoulder which became infected in this case.

The only concrete method to prove whether or not the patient is a true diabetic is to perform the sugar tolerance test, which will prove either negative or positive. In view of the early normal blood sugar report, there is still a doubt in my mind about the nondiabetic element in this case. The old adage, "too many cooks spoil the broth," may be well applied in this case, as Dr. Mackenzie enumerated 14 consultants called to see this patient. It is almost beyond comprehension that they neglected to do a few more blood sugars in order to verify the diabetic hunch that Dr. Mackenzie so ably described.

Relative to the improvement of the patient after the administration of insulin, I would like to refer you to a paper which I recently published in the Annals of Otology, Rhinology and Laryngology, Dec., 1930, on "The Use of Insulin in Postoperative Mastoiditis with Prolonged Drainage."

DR. MACKENZIE (in closing): First of all, I want to thank the several gentlemen for their free discussion of my paper. I am not sure that there will be enough time to answer all the questions that were raised.

Dr. Shemeley touched on a phase of the subject that was rather delicate for me to handle. There is no doubt about my being hampered in the handling of the case by the pathologist up until the time the patient's brother recognized the situation and readily acceded to my request for a consultation with a second pathologist.

Speaking of facial paralysis following operation, we should not be too ready to accept the blame and inwardly accuse ourselves of having committed a blunder at the time of operation, for more often it is due to the extention of the destructive process following the operation and in spite of it. The cause of the facial involvement is to be found rather in one of the dyscrasias, among which must be considered diabetes, syphilis, tuberculosis and a heavy focal infection.

Dr. Shuster's question is a perfectly legitimate one and deserves consideration. It would seem likely that the infection was responsible for the high blood sugar since it is being recognized more and more that infections are responsible for diabetes, the elimination of which is followed by a decrease in the blood sugar to the normal, but most of such improvements have been observed in cases of chronic focal infection; in other words, less active infections than was present in this case. Another reason for suspecting the infection in the mastoid in my case as responsible for the high blood sugar is the

apparent increase of blood sugar. Please remember, however, that the patient had the severe infection in the mastoid for one week before he was operated. Remember, too, that the destruction in the mastoid was progressive until the patient received treatment for his diabetes. I suspected diabetes from the beginning and so told the physicians at the time I was first called in. The laboratory report of 111 m.g. I doubted and therefore ordered and endorsed my requisition for a blood sugar test. The first pathologist claimed it was not necessary because of the absence of sugar in the urine. I am convinced at this time, as I was then, that the original estimate of 111 m.g. of blood sugar was a mistake.

Dr. Stout hit the nail on the head when he said we must not depend too much upon one examination of the blood. It is better to have several tests made before arriving at a final diagnosis. Most of us have experienced at times a sense that a particular something was wrong with a case, based upon the clinical aspects, and when the first report from the laboratory did not confirm our suspicion, it was often confirmed after a second, third or fourth examination.

In reply to Dr. Ersner concerning the Tobey test, the Tobey test is one to be applied in the cases of bilateral middle ear suppuration in which the otologist is in doubt as to which side is involved with an infected thrombophlebitis. But in the case I reported there was no question as to the side. There was a unilateral mastoiditis, pure and simple. Every thinkable test was ordered in this case from the very start, as in our practice in all cases. A further proof that this patient's unfavorable turn was due to the existence of diabetes was the fact that there was a progression of the suppurative process up to the time he was put on an antidiabetic diet, when improvement followed promptly.

As to the administration of insulin in the treatment of obstinate cases of mastoid suppuration, I believe it is a good practice, but only in those cases in which there is definitely high blood sugar, which is controllable in no other way and in which the diabetes is particularly responsible. The treatment of each case depends upon the particular complication that is interfering with recovery. In other words, our diagnosis of each case should be most complete, taking into consideration all etiological factors; furthermore, whether they are still existent or not; all local factors and just how far a particular local factor might be acting as an obstruction to the egress of pus and the normal healing process, also the presence of any dyscrasia. The diet, and all the other things touched upon in the introductory remarks of my paper, must be looked into and corrected, according to the findings.

The appearance of the wound in my case was distinctly that of what I have come to consider as classical for diabetes. However, I would not exclude other factors without confirmatory laboratory findings.

Case Presentation: Bilateral Paralysis of the Facial Nerve, Involving the Auditory Nerve, With Recovery. Dr. P. S. Stout.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. MACKENZIE: The earlier part of Dr. Stout's report suggested the diagnosis of polineuritis cerebralaris meniereformis of Frankl-Hochwart, a condition that is somewhat rare and interesting. I have seen but four cases during the last 25 years. A case of the kind was recently reported by Dr. Howard S. Busler at a staff meeting of the Bryn Mawr Hospital and published in the June issue of the *Eye, Ear, Nose and Throat Monthly*. One of the first cases I saw occurred in a congenital syphilitic patient. This case was reported along with some others but at the moment I am unable to recall where it was published.

My opinion as to diagnosis in Dr. Stout's case changed as he proceeded with the fuller report. The case as I see it is one of straight syphilitic basilar meningitis in which most of the cranial nerves were involved. The membranes of the brain, as you remember, follow certain of the nerves, particularly the second. Furthermore, other of the cranial nerves are attacked as they enter the brain stem, by reason of the inflammatory exudate at this

place, so that in the more severe forms of syphilitic meningitis such a combination of symptoms as presented in Dr. Stout's case do occur.

The fact that the patient had a +4 Wassermann and that it improved under antisyphilitic treatment practically clinched the diagnosis from the therapeutic standpoint.

That the patient had no dizziness is to be explained by the presence of meningitis involving the eighth nerve on both sides, creating thereby a perfect balance. Had the lesion been unlilateral there would have been an imbalance of the labyrinthine tonus on the two sides, when the vertigo would have been pronounced.

In passing I might mention the fact that two entirely different forms of syphilis may produce bilateral deafness. It is the galvanic test which helps us to differentiate them. In the acquired form of syphilis there is meningitis present which extends along the course of the eighth nerve. For this reason the galvanic test is negative. The tardy form of syphilis, on the other hand, selects the inner ear, leaving the nerve intact. In this case though there may be completely negative reactions to turning and the caloric tests, the galvanic reaction remains normal on the two sides.

Summing everything up in this case, there is no doubt, in my mind at least, that the case is one of syphilitic basilar meningitis.

In reply to Dr. Macfarlan, I thought I had made it quite clear in my discussion. To begin with, there was a bilateral involvement of the seventh and eighth nerves, as reported by Dr. Stout. That there should be bilateral involvement is not difficult to understand, for characteristic of generalized syphilis, the lesions are bilateral, in contradistinction to the unilateral lesions of tertiary syphilis. Furthermore, it must be borne in mind that the entrance of these two nerves into the brain stem on the two sides is not separated by more than an inch or an inch-and-a-quarter.

As to lethargic encephalitis: The lesion is in an entirely different location. Lethargic encephalitis is an inflammatory process involving the gray matter surrounding the ventricles and the aqueductus Sylvii. The involvement is necessarily nuclear. As I recall it, there was a convergence weakness. If this condition is present in the absence of other ocular eye muscle involvement the suspicion points strongly toward encephalitis, but in Dr. Stout's case there was involvement of other eye muscles, notably the abducens.

DR. SILVERBERG: I saw this case at the Graduate Hospital and remember distinctly that I was very much impressed by the lethargic appearance of the patient. This immediately suggested an encephalitis to me.

The bilateral involvement could thus be explained very nicely on a basis of bilateral increased intracranial pressure causing pressure changes on both eighth nerves.

The fact that she cleared up so quickly may be explained by the spinal tap or whatever else caused the subsequent decrease in intracranial tension. It certainly is conceivable that this entire picture could have been inflammatory in nature, such as an encephalitic condition, and that the positive Wassermann here was just a coincidence.

DR. MACFARLAN: This case was one of interest from the point of view of the severity of the involvement, and from the rapidity of the recovery. She was so deaf that she could not hear words shouted in her ears; she showed no response by A. C. or B. C. to a large middle range fork. There was no caloric reaction, no past-pointing, no dizziness. Both facials were apparently totally paralyzed.

One of the Graduate School men very wisely observed the extreme dullness that suggested encephalitis lethargica. One expects some dullness in extreme deafness; however, she seemed to have too much dullness.

Just two nerves seemed involved, the seventh and eighth.

Dr. Stout mentions that there was a subjective sensation of objects moving slightly, after turning. Often when the patient opens the eyes while turning there is a visual confusion on stopping. This is a purely ocular phenomenon and can occur independently of the vestibular status. It is analagous to the auditory after image of sound.

Her vestibular loss must have been progressively equal on both sides, else there would have been imbalance and dizziness. Her hearing was equally lost.

What is back of this thing is certainly a mystery, particularly so when she cleared up so quickly.

I would like to ask Dr. Mackenzie how he can conceive of a local meningeal involvement that could be so absolutely symmetrical and involve just these two nerves on each side.

Some Findings in Paracusis. Dr. Douglass Macfarlan.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. SHUSTER: I have tested about 1,000 children with the phonograph audiometer and found that extraneous noise interferes with the test alike, with the normal children as well as the deafened.

DR. MACFARLAN (in closing): Dr. Shuster brings up the point that on account of the paracusis idea, many charlatans are using noise producers in an attempt to relieve deafness and tinnitus. They are exploiting the paracusis idea. I would emphatically say that noise producers do not cure deafness. In testing hearing before and after use, the deafness is found to be somewhat increased. When any noises cease there comes the consciousness of hearing improvement, but the comparison is with the hearing when the noise was on, not with the hearing prior to the noise.

As to audiometer testing in schools, this test cannot be well done by anyone, as suggested in the advertising. One must know that outside noise and distraction affect the child's record. Unfortunately, the noises are apt to occur when the word is being spoken from the record, and more unfortunately, the child is given only one test word at each intensity in each of four columns.

I prefer a constant intensity record, set at a threshold at which normal children should hear; else I prefer a buzzer audiometer. Either of these methods interfere less with the school curriculum and can be made a part of the routine physical examination.

The Comparative Surgical Anatomy of Infant, Child and Adult Mastoids. Dr. P. S. Stout.

(Appears in full in this issue of THE LARYNGOSCOPE.*)*

THE NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of March 16, 1931.

Dr. Hilliard Wood, Chairman, Presiding.

A Case of Mastoid Infection. Dr. M. M. Cullom.

Dr. Cullom had been called by Miss J., on Feb. 12, 1931, on account of great pain in the left ear for three days. There was a history of an attack of influenza. Examination: There was extreme tenderness over the left mastoid. The eardrum was red and bulging. X-ray of the mastoid by Dr. Floyd on Feb. 23 revealed involvement of all the mastoid cells.

Treatment: The drum was incised and a profuse discharge of bloody pus ensued.

The condition continued to grow worse and at the end of one week the temperature was 103° and the patient suffered acute pain. An immediate operation was advised.

Dr. Kennon was called in consultation. At the time the patient was seen by him the tenderness over the mastoid had decreased, the discharge had diminished, and the temperature had fallen to normal. It was considered that the cells had broken down and that the process was localizing with a limiting membrane. The patient complained of neuralgia in the left temple and of a throbbing sensation in the left mastoid. Dr. Kennon advised against operation. At his request a second X-ray examination was made, which showed possibly some more resolution of the mastoid cells.

Dr. Cullom stated that it was his theory that most mastoids were caused by purulent infection of the nasal accessory, the X-ray in this case showed dense clouding of the right antrum, and Dr. Cullom remarked that this was the first time that the sinus was on the side opposite the mastoid involved. He also brought out that the infection of the sinus may have cleared up so that at the time of development of mastoiditis that involvement of the sinus could not be proven.

DISCUSSION.

Dr. W. W. Wilkerson said that this paper reminded him of a patient, age 8 years, who had been under his observance recently. The child had had influenza and measles several weeks before he saw him. There was profuse discharge from the ear and some drooping. X-ray showed complete breaking down of all mastoid cells. Due to the fact that the patient was just recovering from measles, it was decided to wait on the mastoidectomy for a few days. Two weeks later his ear had ceased to discharge; the drum was still bulging, no redness and no drooping. A second X-ray examination showed that there was no change in the condition. Dr. Wilkerson had not seen the patient since, but Dr. Kennon, in consultation, had reported that there was no mastoid tenderness and no fever; hearing was normal, and that the drum was entirely normal, and that all the drooping had entirely disappeared. Dr. Wilkerson thought that the case was very interesting, as it provoked much argument as to when to operate.

Dr. E. L. Roberts said that he thought that it was better to operate when there were sufficient positive findings, rather than not to operate. He felt that many of the chronic cases would be better off if they had had surgery in the acute stages.

Dr. Hilliard Wood said that he thought that the best time to operate on a mastoid was when the abscess was ripe. He thought that it would get well faster than if done in the incipient stage. He thought the history, the time it had been running, the W. B. C., tenderness on deep pressure and the X-ray picture, if interpreted in the light of all the findings, were of a great help.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. Harold Walker, 5 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Briarcliff Lodge, Briarcliff Manor, Westchester County, New York, June 18-20, 1931.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Atlantic City, N. J., June 15-17, 1931.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63rd St., New York.
Place: St. Louis, June 3-4-5, 1931.

AMERICAN BRONCHOSCOPIC SOCIETY.

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.
Place: St. Louis, June 6, 1931.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Indiana, Sept. 14-19, 1931.

IN MEMORIAM

One of the saddest calamities that has befallen American Laryngology was the sudden death by automobile accident on the Dixie Highway near Richmond, Ky., of two of our most distinguished colleagues, Dr. Joseph Addison Stucky, of Lexington, Ky., and Dr. Robert Clyde Lynch, of New Orleans, La.

Had I been asked to name the two most eminent and most popular oto-laryngologists of the Southland, I would unhesitatingly have named these dear friends whose untimely death must be accounted not only a sad and serious loss to the communities in which they lived and labored and to their hosts of friends throughout the country, but also to the national community and to the medical profession at large.

Leaders in their field, distinguished in oto-laryngology, forceful in their personalities; both lived and worked actively, effectively and generously; both will be mourned by hosts of friends; both will be remembered by many kindly acts, by many contributions of real merit to medical science, by lives worthy of emulation.

JOSEPH ADDISON STUCKY.

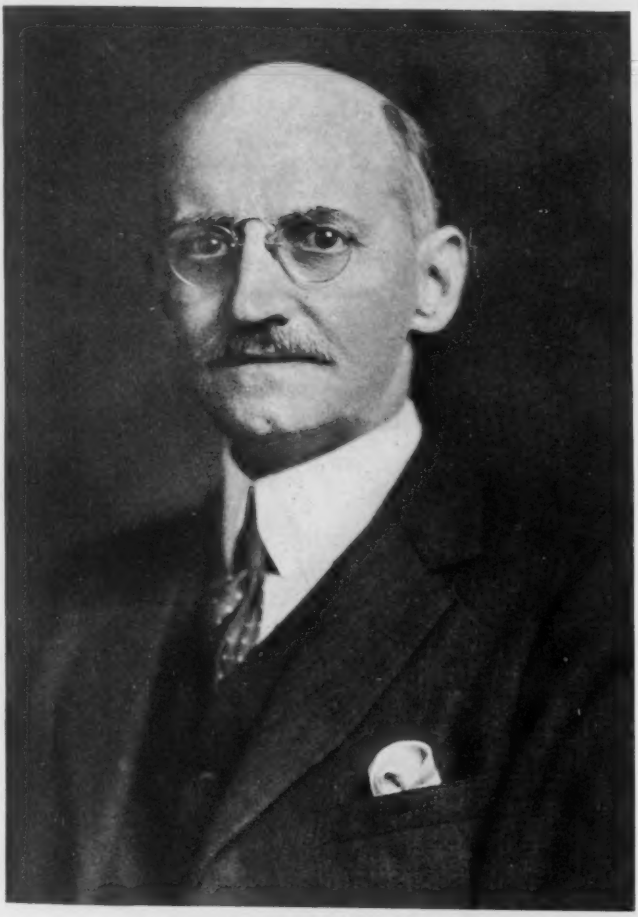
Joseph Addison Stucky was born in Louisville, Sept. 6, 1857. His great-grandfather, John Stucky, was one of the first settlers in that part of the state. After attending the public schools of Louisville, Dr. Stucky entered the University of Louisville, graduating from the College of Medicine in 1878. Following his graduation he moved to Lexington and began the practice of medicine. For nine years he was a general practitioner, but since that time devoted himself exclusively to ophthalmology and otolaryngology. In his long and faithful service to the specialties of medicine he gave of his time, his energies, his talents, as much as man could give.

One of the outstanding efforts in Dr. Stucky's long and distinguished career was the establishment of special clinics in the Kentucky mountains for the treatment of Trachoma and operative surgery in Eye, Ear, Nose and Throat work. These clinics, the first to be established in the mountains more than a quarter of a century ago, had grown to such proportion that hundreds of mountain people were treated yearly by Dr. Stucky and his associates.

As one writer eloquently puts it: "No man gave more of himself than he did. In the valleys and coves of the mountains there are men, women and children whose lives have been made richer and fuller and freer because of him, who have been relieved of bodily ills and given health and strength and sight because he gave so generously of himself and of his skill.

"Never opportunity came to him to speak a kind word or do a helpful act of which he did not take fullest advantage.

"Past the allotted span of life, he was still vibrant with undimmed interest in every subject, important or trivial, that affected those for



JOSEPH ADDISON STUCKY

whom he cared or the community in which he played for so many years so potent a part.

"His associates will speak of his professional attainments; will tell of the national recognition of his ability and skill. Great, however, as was his skill, notable as was his ability in his chosen and beloved profession, even greater was his capacity for friendship, his passion for service."

Dr. Stucky was a former President of the American Academy of Ophthalmology and Otolaryngology and of the American Laryngological, Rhinological and Otological Society. It was this latter organization that was dearest to his heart, as every Fellow who has heard his eloquence at annual meetings of this organization will testify.

His almost prophetic vision and sound perspective in our science, together with a very bountiful supply of common sense, characterized the quality and the substance of his various scientific contributions and participation in discussions.

What a dear and loyal friend he was! How he basked in the sunshine of his friendships! To the very last he was visiting with a dear friend and was preparing for a gala visit to meet many more friends in St. Louis on the occasion of the annual meeting of his most beloved society.

To his bereaved family we offer our deepest sympathy and assurance that the memory of his friendship and of his kindliness of heart will endure. M. A. G.

ROBERT CLYDE LYNCH.

Robert Clyde Lynch, son of Dr. William and Minerva Maitlen Lynch, was born in Carson City, Nev., Sept. 8, 1880. When he was three years of age his family moved to New Orleans and he received his early education in the public schools there, graduating from Tulane University Medical School in 1903. After practicing general medicine for two years in Natchitoches, he studied the specialties of Ophthalmology and Otolaryngology, first in New Orleans, until August, 1906, and then in London, Paris, Vienna and Freiburg, until 1907.

Early in his career Dr. Lynch was associated with the late Dr. A. W. DeRoaldes and on the death of the latter was named Surgeon in Charge and in 1930, Surgeon in Chief of the New Orleans, Eye, Ear, Nose and Throat Hospital. He was Professor of Otolaryngology in the Post-Graduate School of Medicine of Tulane University and Consultant in Otolaryngology in the Touro Infirmary. For several years he had served as a member of the Editorial Board of the Archives of Otolaryngology and as a collaborator on several other medical journals. He was a member of the New Orleans local and Louisiana State Medical Associations, the American Medical Association, the American College of Surgeons, and the American Academy of Ophthalmology and Otolaryngology; President (1924) of the American Bronchoscopic Society and a Fellow of the American Laryngological Association and of the American Laryngological, Rhinological and Otological Society.

He is survived by his widow, Amanda Genin Lynch, and by two sons, Mercer Genin Lynch and Robert Clyde Lynch, Jr.

Outstanding among his professional accomplishments as a Laryngologist especial mention must be made of his brilliant surgery, wide

and varied experience and unusually successful results in Malignant Neoplasms of the Larynx. Many of these operations were done under suspension laryngoscopy with instruments and apparatus which he had devised and perfected. As a clinician and special surgeon and as a lucid and forceful teacher in Laryngology, he achieved national recognition and distinction. He was equally distinguished for his kindliness of nature, his charitable service and for his thoughtfulness toward his friends.

He was only 52 years of age, was in the most useful period of his splendid professional career and had the respect and love of his colleagues wherever he went.

Pre-eminent among his contributions to Otolaryngologic literature are the following:

1915—Technique of Suspension Laryngoscopy. Read before Section of Laryngology and Rhinology, New York Academy of Medicine, May 25, 1915. THE LARYNGOSCOPE, Dec., 1915.

1915—Suspension Laryngoscopy and Its Accomplishments. Candidate's Thesis to the American Laryngological Society. Annals of Otology, Rhinology and Laryngology, Sept., 1915.

1916—Resumé of My Year's Work with Suspension Laryngoscopy. Read before the American Laryngological Association, Washington, D. C., May 10, 1916. THE LARYNGOSCOPE, St. Louis, Dec., 1916.

1916—The Possibilities and Limitations of Suspension Laryngoscopy. Read before Section, Laryngology, Otology and Rhinology at 67th Annual Session of A. M. A., Detroit, June, 1916. Journal of American Medical Association, Jan. 27, 1917.

1916—Suspension in Bronchoscopy and Esophagoscopy. Read before Clinical Congress of Surgeons of North America in Philadelphia, Oct. 25, 1916. Surgery, Gynecology and Obstetrics, Aug., 1917.

1918—Some Bronchoscopic and Esophagoscopic Foreign Bodies I Have Known. Read before the Section of Ophthalmology, Otology, Rhinology and Laryngology at State Medical Association of Texas, San Antonio. Texas State Journal of Medicine, Dec., 1918.

1918—Suspension as I Use It Today. Read at American Laryngological Society, May, 1918. The Southern Medical Journal, Jan., 1919.

1920 Fluoroscope Bronchoscopy, Esophagoscopy and Gastroscopy. Read before American Laryngological Association in Boston, May 28, 1920. THE LARYNGOSCOPE, St. Louis, Nov., 1920.

1920—Fluoroscopic Bronchoscopy, Esophagoscopy and Gastroscopy. Read at Third Annual Meeting of Association of American Peroral Endoscopists at Boston, June, 1920. New York Medical Jour., March 16, 1921.

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1920—The Technique of Radical Frontal Sinus Operation Which Has Given Me the Best Results. Read before the Twenty-sixth Annual Meeting of the American Triological Society at Boston, June 2, 1920. THE LARYNGOSCOPE, St. Louis, Jan., 1921.

1921—Suspension Laryngoscopy and Its Problems. Written for the occasion of the twenty-fifth birthday of THE LARYNGOSCOPE. THE LARYNGOSCOPE, St. Louis, July, 1921.

1923—Technique of a Pansinus Operation. Read in Section, Eye, Ear, Nose and Throat of Southern Medical Association, Seventeenth Annual Meeting at Washington, D. C., Nov. 12-15, 1923. Southern Medical Jour., April, 1924.

1923—Why We Should Demand Poison Labels On Concentrated Lye and Washing Powder Packages. Jour. of Florida Medical Association, Nov., 1923.

1923—Diagnosis of Strictures of the Esophagus. Read before the Louisiana State Medical Society on April 24-26, 1923. New Orleans Medical and Surgical Jour., Dec., 1923.

1924—Pot-Pourri, President's Address, American Bronchoscopic Society, St. Louis, May 28, 1924. THE LARYNGOSCOPE, St. Louis, Oct., 1924.

1926—Some Otolaryngological Problems, Chairman's Address, Section of Eye, Ear, Nose and Throat, Southern Medical Association, Twentieth Annual Meeting, Atlanta, Ga., Nov. 15-18, 1926. Southern Medical Jour., Dec., 1926.

1926—Cancer of the Larynx. Transactions of the Indiana Academy of Ophthalmology and Otolaryngology, Jan., 1926.

1928—Stenosis of the Larynx. Transactions of the American Academy of Ophthalmology and Otolaryngology, Jan., 1928.

1929—Mixed Tumor of the Parotid Type, Growing from the Posterior Aspect of the Thyroid Cartilage. Read at the Twelfth Annual Meeting of American Bronchoscopic Society, San Francisco, July 6, 1929. Archives of Otolaryngology, May, 1930.

1929—Endothelioma of the Larynx. Read before the American Larynogological Association. Transactions of the American Laryngological Association, 1929.

1929—Sinus Disease in Infants and Children. Read before the Louisiana State Medical Society, April 9-11, 1929. New Orleans Medical and Surgical Jour., Dec., 1929.

1929—Symposium On Ethmoidal Sinusitis, External Radical Approach. Read before the American Academy of Ophthalmology and Otolaryngology. Transactions of the American Academy of Ophthalmology and Otolaryngology, 1929.

1930—Sinus Diseases in Children. Read before Section, Eye, Ear, Nose and Throat at Sixty-third Annual Session, Mississippi State Medical Association, Vicksburg, Miss., May 14, 1930. New Orleans Medical and Surgical Jour., Sept., 1930.

To dear Mrs. Lynch and the boys we offer our heartfelt sympathy; they have lost a precious loved one, we have lost a real friend and highly esteemed colleague. M. A. G.